C.V.S

# Cardiovascular diseases

## *Introduction*

- Cardiovascular diseases are the most common cause of death worldwide.
- CVD are the most common cause of death in MALES & FEMALES.

## *Anatomy*

- 2 Atria and 2 Ventricles
- The Rt atrium & ventricle lie anteriorly while Lt atrium & ventricle lie posteriorly.
- Most anterior chamber in the heart is Rt ventricle and its immediately under the sternum so right ventricle enlargement will → Left Parasternal heave.
- Most Posterior chamber in the heart is Left atrium.
- Mitral valve is the only bicuspid valve. Tricuspid, Aortic, & Pulmonic valves are tricuspid

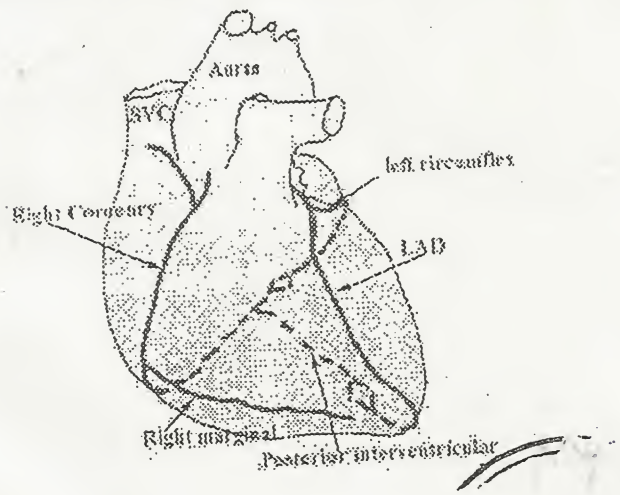
## *Coronary circulation*

Aorta gives rise to:

1. Left coronary artery (LCA) which gives the following branches:
   - left anterior descending artery (LAD)—supplies→ Anteroseptal wall & Apex
   - left circumflex artery (LCX) –supplies→ Lateral wall
     - In 10% of pts LCX gives Posterior descending artery –supplies→ Posterior wall
2. Right coronary artery (RCA) which gives the following branches:
   - Marginal branch—supplies→ Inferior wall
   - Posterior descending artery (PDA) in 90% of pts—supplies→ Posterior wall
   - Sinoatrial (SA) nodal artery in 60% of individuals
   - **Atrioventricular (AV) nodal artery in 90% of individuals**

Coronary artery dominance: It depends on the artery that supplies the posterior descending artery (PDA). If it arises from RCA (90% of individuals) then it's called dominant right system circulation and if it arises from the CX (10%) of individuals then it's called dominant left system.

| MI region | Artery occluded |
|---|---|
| Anteroseptal | LAD |
| Lateral | LCX |
| Anterolateral | LCA |
| Inferior | RCA marginal |
| Posterior | RCA or LCX |



## ✡ *Nerve supply of the heart* ✡

- Sympathetic innervations acts on **β1 receptors**:
  1. To the myocardium of both atria & ventricles → +ve inotropic effect = ↑ power of contraction
  2. To SA → +ve chronotropic effect = ↑ heart rate
  3. To AV nodes → ↓ AV delay = ↑ conduction

- Parasympathetic innervations from the vagal nerve acts on $M_2$ receptors:

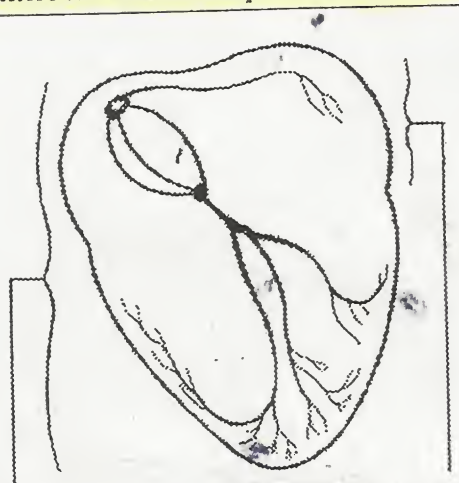
1. It **does not** supply the myocardium of the ventricles → **no effect on contraction** [it supplies only the atrial myocardium]
2. To SA → -ve chronotropic effect= ↓ **heart rate**
3. To AV nodes → ↑ AV delay = ↓ conduction

Normally heart rate normally changes according to the body need for example it increases in exercise or infection also it changes with respiration **but** in pts with **Autonomic Neuropathy** [e.g. **DM**] these normal changes are decreased.

Cardiac Parasympathetic supply (Vagus ) can be stimulated by some maneuvers:

1. Carotid artery massage
2. Valsalva maneuver: forcibly expiration against a closed airway.

**These maneuvers are important in Rx of Supraventricular arrhythmias**



SYMPATHETIC

- Innervates both atria and ventricles
- Increases
  - Heart Rate
  - AV Conduction
  - Irritability

PARASYMPATHETIC

- Innervates the atria via vagus nerve
- Little or no effect on function of ventricles
- Decreases
  - Heart Rate
  - AV Conduction
  - Irritability

## *Conducting system of the heart*

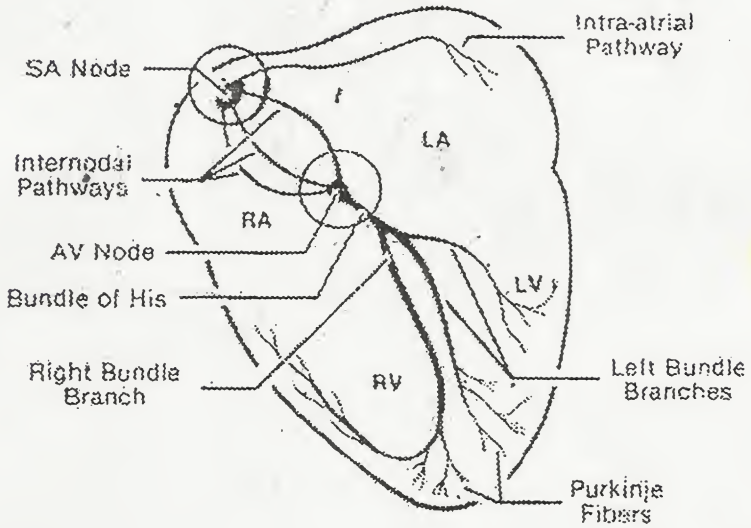
SA node generates an impulse → reaches both atria & AV node → contraction of both atria & **AV node delays the impulse** transmission to the ventricle until the atria complete their contraction → impulse travels from AV node to the bundle of His →to the Right and Left bundles → to Purkinje network → Ventricular contraction.

SA node location: at the junction of the superior vena cava and right atrium.
AV node location: in the right atrium the lower end of the interatrial septum.

Note: Direct conduction between atria &ventricles is prevented by the annulus fibrosus (fibrous ring) & is **traversed ONLY by the bundle of His in normal hearts.**

Q. What controls the heart rate ?
Heart rate is controlled by the fastest pacemaker = rate maker.
Normally the fastest pacemaker is the **SA node** with **70 beats/min**, if the SA node is blocked then *escape rhythm* arise in the lower conducting system the → **AV node 60 beats/min** if AV node is blocked → then **His bundle** with **50 beats/min, OR Purkinje** fibers with **25-45 beats/min**.

They are called escape rhythm because they escape from normal inhibition by SAN.



## *Physiology of the CVS*

Definitions:

- End-systolic volume: volume of the blood in the heart at the end of systole
- End-diastolic volume: volume of the blood in the heart at the end of diastole
- Stroke volume = End-diastolic volume - End-systolic volume
- Cardiac Output = Stroke volume × Heart rate and is normally 5 liters/minute
- Ejection Fraction: Stroke volume / End-diastolic volume **[normally > 0.55 = 55%]**

➢ **Preload:** is End-diastolic volume = the degree of stretching of cardiac muscle cells before contraction [when venous return ↑ → End-diastolic volume ↑ → stretches the ventricular muscle fiber (Starling law) → ↑ the force of contraction = ↑ in Oxygen demand]

*Factors affecting preload:*

1- The **tone of the venous system.**
¾ of blood in the body is in the venules [capacitance vessels] of venous system SO:
If drug or neurotransmitter (e.g. Ang II) causes venoconstriction → ↑ venous return → ↑ EDV → ↑ Preload **and if** a drug (e.g. ACEI) or neurotransmitter causes venodilatation →↓ venous return → ↓ EDV → ↓ Preload

2- The **volume of Extracellular fluid** which depends on amount of Na reabsorption by the kidney so if a substance (aldesterone) is overproduced → Na reabsorption by the kidney → ↑ preload **and if** Drug (diuretic) is used or a Natriuretic hormone (ANP) is produced it will ↓ Na reabsorption by kidney → ↓ in the Preload

➢ **Afterload:** pressure that must be overcome for ventricles to eject blood from heart, so for the left vent. is Aortic pressure and for right vent. It is Pulmonary pressure.

*Factors affecting afterload:*

The aortic pressure is depends on the degree of vasoconstriction of arterioles [resistance vessels] SO:

1-If a vasoconstrictor substance (e.g. noradrenalin) is overproduced it will→ ↑ arteriolar contraction which will→ ↑ Afterload→ force of contraction→ ↑ $O_2$ demand.

2-If a vasodilator substance (e.g. calcium channel blocker) is used it will → ↓ arteriolar contraction → ↓ Afterload→ ↓ force of contraction→ ↓ $O_2$ demand.

Factors Affecting Cardiac output [CO = SV X HR]

1. ↑ Heart rate → ↑ CO
2. ↑ Preload → ↑ CO
3. ↑ Afterload → ↓ CO
4. ↑ Contractility → ↑ CO

# Heart Failure

## *Definition*

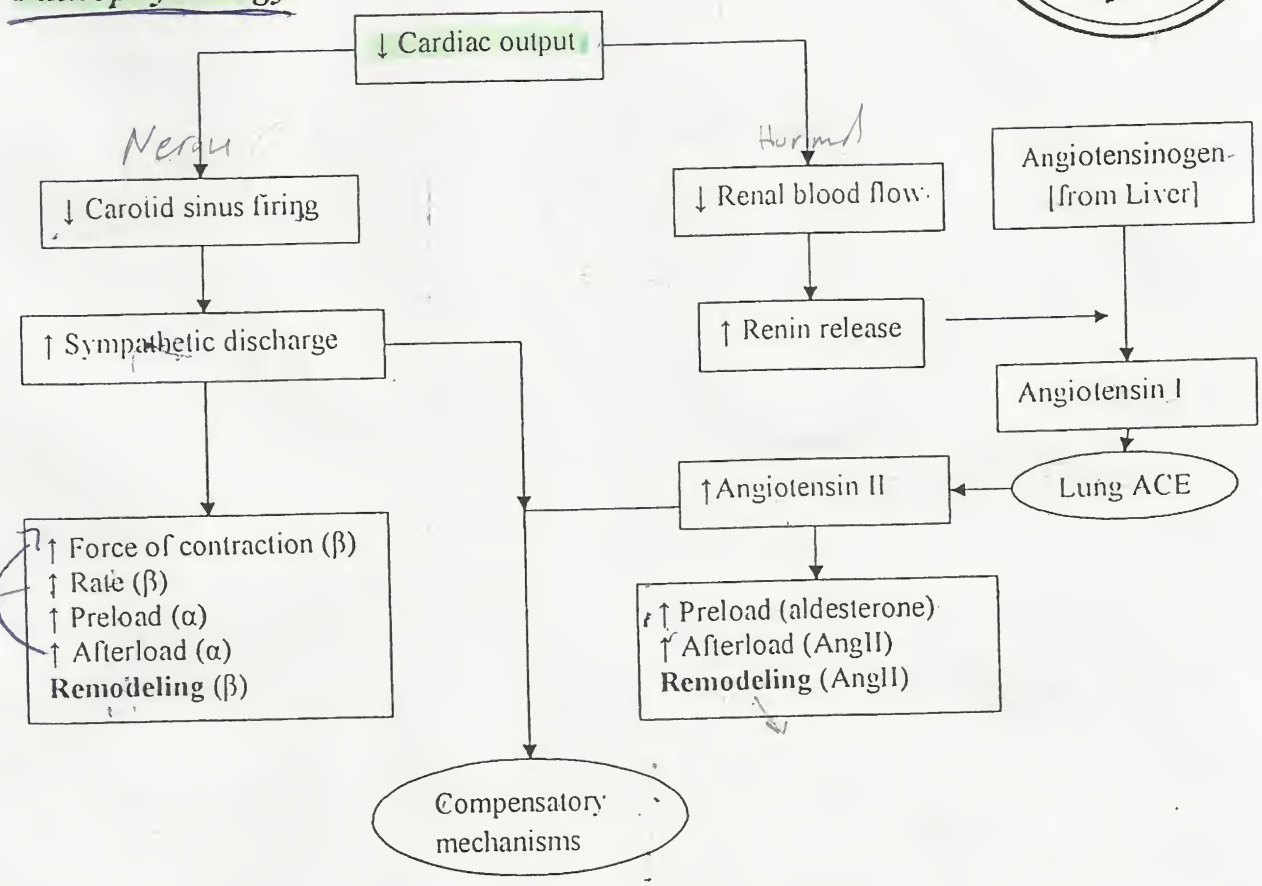
**It is a clinical syndrome** in which the patient is having symptoms and signs of heart failure in the presence of a cardiac abnormality.



## *Epidemiology*

- It affects 2% of adult population.
- It affects 10% of those aged ≥75 years.

## *Pathophysiology*




If these compensatory mechanism maintain the cardiac output and pt remains asymptomatic it is called [**Compensated heart failure**] but when these mechanisms fail and the pt becomes symptomatic it is called [**Uncompensated heart failure**].

*Ventricular Remodeling:*

**Definition**: ventricular remodeling are changes in the size, and configuration of the ventricle as a consequence of hemodynamic changes due to decreased Cardiac output

- Remodeling is triggered by substances as **Catecholamines** & **Angiotensin II.**
- Changes in myocyte during remodeling will cause reduced effectiveness of ejection

## *Etiology* [ANY DISEASE IN THE HEART CAN CAUSE HAERT FAILURE]

1. Ischemic heart disease [**Most common cause**]
2. Hypertension
3. Cardiomyopathies
4. Valvular heart disease

➢ High out-put heart failure:

1. Thyrotoxicosis
2. Anemia
3. Arteriovenous shunts
4. Beriberi

Note: The most common cause of RVF is LVF.

### *Classifications of heart failure*

According to chamber involved
- Left ventricular failure [LVF]
- Right ventricular failure [RVF]
- Biventricular failure = Congestive heart failure

According to the clinical picture
- Acute heart failure
- Chronic heart failure

According to cardiac cycle
- Systolic heart failure [Ejection fraction < 40 %] e.g. IHD, HTN
- Diastolic heart failure [Ejection fraction ~~< 40 %~~ normal] e.g. HTN & some cardiomyopathies

## *Clinical picture*

### ➢ Symptoms due to pulmonary venous congestion (LVF):

*1. Breathlessness (Dyspnea)*

**Definition**: its abnormal awareness of breathing.

Pathophysiology:

- LVF → ↑ in hydrostatic pressure in the pulmonary vessels → movement of fluid to interstitial space [**interstitial edema**] → ↓ the compliance of lungs → ↑ work of breathing→ dyspnea.
- Further elevation of in pressure → disruption of tight junction between the alveolar cells with movement of fluids into the alveolar space [**alveolar edema**]

**Cough** may occur → when due to interstitial edema it's dry but when due to alveolar edema it becomes productive of white to pink sputum (pulm. Edema)

*2. Orthopnea*

**Definition**: breathlessness on lying down.

Pathophysiology:

Due to redistribution of fluid from the abdomen and lower extremities into chest during lying flat → ↑ pulmonary hydrostatic pressure → see as above.

*3. Paroxysmal Nocturnal Dyspnea [PND]* → Cardiac Asthma 2-3 hrs

**Definition**: sudden breathlessness after period of sleep.

It's also know as *Cardiac asthma* due to presence of wheeze but the narrowing of bronchioles here is due to edema and congestion of bronchioles.

### ➢ Symptoms due to reduced cardiac output (LVF):

1- Fatigue & weakness→ due to reduce blood supply to the muscle

2- **Nocturia** →

- During the day, when the skeletal muscles are active, the limited cardiac output is shifted away from the kidney toward the skeletal muscles. The kidney interprets this decrease in blood flow as hypovolemia → activation of the RAAS [Renin-Angiotensin-Aldesterone-system].
- At night, when the pt is at rest, cardiac output is shifted back to the kidney & diuresis occurs. [BED REST IS A DIURETIC FOR PT WITH HEART FAILURE]

2- **Cheyne-Stoke breathing** (Periodic breathing)

It is periods of apnea alternates with periods of rapid, heavy breathing (**hyperpnea**)

3- **Cerebral symptoms**→ due to ↓ perfusion and hypoxemia patient may develop confusion, difficulty in concentration, headache, & insomnia.

### ➢ Symptoms due to systemic venous congestion (RVF):

- **Anorexia** and nausea due to liver congestion.
- Congestion of GIT → malabsorption → [Cardiac cachexia] rare.

## Signs

**LVF →↓** peripheral perfusion:

1- Activation of sympathetic system → **Tachycardia, & cold sweaty extremities** [If Hypotension then the pt is in SHOCK]
2- **Pulsus alternans** which is regular rhythm with alternating strong and weak contraction. **It's a sign of sever HF** → Poor prognosis
3- ↑ Pulmonary congestion → **Bilateral basal crepitations,** or **Pleural effusion** [heart failure is the most common cause for transudative PE]

Pulmonary Edema

**RVF →** ↑ in systemic venous pressure leading to:

1- **Raised JVP**
2- Cardiac edema:
   - ↑ hydrostatic pressure in the venous system will cause fluid to move from intravascular volume to the interstitial space
   - The edema is dependent so in ambulatory pt it occurs in the lower limb "**ankle edema**" & in bed ridden pt it occurs over the sacrum "**Sacral edema**"
3- **Congestive Hepatomegaly:** it is painful (due to distension of Gilson's capsule), & this liver congestion may result in **Jaundice** with elevated liver enzymes

**Precordial examination**

1- May find sign of MR or TR due to ventricular dilatation
2- S3, & S4 occur in heart failure

| Right Vent. Failure | Left Vent. Failure |
|---|---|
| • LLE ± Ascites<br>• Hepatomegaly ± Jaundice<br>• ↑ JVP | • Dyspnea<br>• Orthopnea<br>• PND<br>• Bilateral basal crepitations |

When the pt changes from compensated state to uncompensated state acutely, look for the precipitating factor for this uncompensation.

**Precipitating factors** are:

1- **Arrhythmias**
2- Infarction
3- Infection, Anemia, Pregnancy, Thyrotoxicosis
4- Infective endocarditis
5- Pulmonary embolism

## *Investigations*

Management of CHF must focus on the cause of the heart failure, not simply on relieving the symptoms because Heart failure is not a diagnosis but it's the common end-result of many pathological processes.



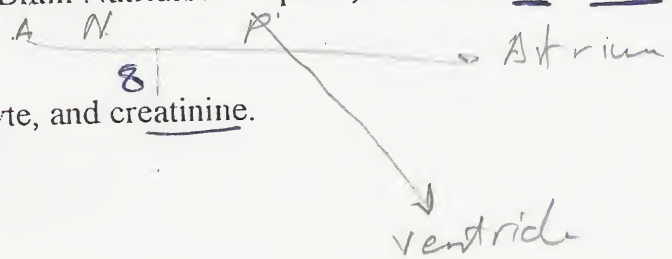
1. **ECG** may show the cause. For example MI, or Arrhythmias

2. **CXR** may show
   - Cardiomegaly
   - Congestion in the lungs:
     - Kerley B line
       - They correspond to the stage of interstitial edema
       - It represent interlobular lymphatics that are distended by fluid
       - Found in periphery of lung bases, 1-2 cm, & are horizontal
     - Acute pulmonary edema
     - Pleural effusion

3. **Echo**: to see the Valves, Chambers, Movement of the wall, & EJECTION FRACTION which is The **most commonly** used indicator of ventricular function & is normally > 55%.

4. Radionuclide ventriculography is used to measure right and left ventricular ejection fraction. [Better than echocardiography in measuring ejection fraction]

5. **Raised BNP** (Brain Natriuretic Peptide): useful in Dx, prognosis, & monitoring of therapy.




6, Urea, electrolyte, and creatinine.

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## Management of Acute Heart Failure→Pulmonary Edema [ER]

1- CALL FOR HELP
2- Sit the pt up [to ↓ venous return]
3- Insert IV line
4- Give **Oxygen**
5- **Morphine** To relive anxiety & dyspnea, and ↓ preload by causing venodilatation
6- IV diuretics [**Furosemide** is the diuretic of choice because it it acts more rapidly than any other diuretic & has a potent **vasodilator** effect on of the systemic arterial and venous systems]
7- **Glyceryl TriNitrate** [GTN] Sublingually first –if fails→ IV
   - Acts predominantly as **venodilators**, and its other action is coronary artery vasodilation
   - Contraindicated in patient with systolic BP should be < 110 mmHg, or pts taking sildenafil.
8- Venesection may be used
9- to improve left ventricular contraction:
   - A) **Digitalis**: Its main use now is control of ventricular rate in patients with rapid atrial fibrillation or flutter and LV dysfunction, because they do not have the negative inotropic effects of other drugs that inhibit atrioventricular nodal conduction [β-blockers & calcium channel blockers].
   - B) Intra-aortic balloon pump

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## *Treatment of Chronic heart failure*

Aims of treatment

- Symptomatic relief. [by reducing the PRELOAD & AFTERLOAD]
- Removal of precipitating factors.
- Control of underlying cardiac disease.
- Improve the prognosis

### ➢ General measures

1- **Bed rest in acute phase**: bed rest is a diuretic in pt with heart failure. (because of bed rest pt with CHF have ↑ risk for thromboembolism, therefore rest is maintained till clinical improvement & after that the pt is encouraged to move)

2- **Diet** → ↑Weight→↑ cardiac work. So overweight pt are advised to ↓ wt.
→ Salt-restriction can reduce the preload.

3- **Stop smoking**

4- **Alcohol abstinence** because Alcohol → ↓ contractility, → ↑ arrhythmias, → May cause Dilated cardiomyopathy

5- **Vaccination** against Influenza & pneumococcus.

### ➢ Drug treatment

**Diuretics**

*Pharmacodynamics*:

- Increase sodium & water excretion → ↓ preload
- Weak arterial and venous dilatation→ ↓ afterload & ↓ preload
- **Loop diuretic (Furosemide)** is the first line treatment (mechanism of action → reversible inhibition of the reabsorption of $Na^+$, $K^+$, and $Cl^-$ in the thick ascending limb of Henle's loop)
- **Thiazides** (metolazone) they work synergistically with loop diuretics (mechanism of action reduce the reabsorption of $Na^+$ and $Cl^-$ in the first half of the distal convoluted tubule)

*Indication in HF*: Loop diuretics or Thiazides are given to all symptomatic patients

*Side effects:*

1- Volume depletion and hyponatremia
2- ↓ hypokalemia with Furosemide or Thiazides [↓K→ ↑ risk of digitalis toxicity]
3- Thiazide [hyperuricemia, hyperglycemia, hypercalcemia].
4- Metabolic alkalosis

*Effect on prognosis:*

They are not shown to improve the prognosis

**Angiotensin-converting enzyme inhibitors (ACEI)** [Captopril, Enalopril, Lisinopril]

*Pharmacodynamics*

- ACE inhibitors mainly act by ↓ afterload by ↓ the formation of AngII.
- ACEI also ↓ preload by ↓ the formation of aldesterone.
- Inhibition of the remodeling.

*Indication in HF*: ACEI are given to all patients with heart failure or having risk factors for heart failure

*Side effects:*

1. Most common side effect is cough [in10-15% of pts], it is due to accumulation of bradykinins which normally is converted to kinin by the ACE enzyme.
2. First dose hypotension
3. Hyperkalemia
4. Rare leukopenia, rash, and angioedema. → absolute contraindication

*Effect on prognosis:*

They have been shown to prolong survival & reduce hospitalization.

*Absolute contraindications* **for ACEI & Angiotensin receptor blockers**

1. Pregnancy
2. Hyperkalemia serum K > 5.5
3. Bilateral renal artery stenosis
4. Advanced renal failure (e.g., creatinine > 3 mg/dL)

Note: Angiotensin receptor blockers [Losartan] is used when the patient is intolerant to cough induced by ACEI.

**β-Blockers**

*Pharmacodynamics:* They act by protecting the myocardium against sympathetic stimulation i.e. inhibiting the remodeling.

*Indication in HF*: N A H

They indicated for patients with NYHA II or III

*Effect on prognosis:*

β-blockers with evidence to prolong survival & reduce hospitalization.:

1. Metoprolol
2. Carvedilol
3. Bisoprolol
4. Bucindolol
5. Nebivolol

*Contraindication*:

A→ Asthma
B→ Bradycardia
C→ Cardiac failure NYHA IV = unstable heart failure
P→ Peripheral vascular disease

hypotension

(4) **Aldesterone antagonist [Spironolactone]** Aldosterone receptor antagonists, improve long-term clinical outcome in patients with severe heart failure [NYHA IV]

**Side effects:**

1. Hyperkalemia
2. Gynecomastia

## Digitalis [Digoxin & Digitoxin]

*Pharmacodynamics*:

- Inhibition of the $Na^+/K^+$ pump leads to increased intracellular $Na^+$ levels, which in turn slow down the extrusion of $Ca^{2+}$ by the $Na^+/Ca^{2+}$ exchange pump → ↑ intracellular $Ca^{2+}$ → ↑ Contractility. +chronotropic +inotropic
- Digoxin also acts on CNS → ↑ vagal activity→ ↓ conduction through the AVN. -chronotropic
- Half-life for digoxin is 40 hours.
- Metabolism → Digoxin by the kidney
  → Digitoxin by the liver

*Effect on prognosis:*

- Digitalis does **not prolong survival but it reduces rehospitalization rate**

*Indication in HF*:

(1) Marked systolic dysfunction.
(2) Heart failure associated with atrial fibrillation and rapid ventricular rate.

*Side effects (Toxicity)*:

**Gastrointestinal** (relate to vagal effects)

- anorexia
- abdominal discomfort / pain
- vomiting
- diarrhea

**Cardiac - any arrhythmia ever described** either atrial or ventricular (e.g. pulsus bigeminy or trigeminy) or heart block

Gynecomastia

Confusion

Yellow color vision (xanthopsia)

**Treatment of Toxicity**

- Stop giving the drug
- Antiarrhythmics (lidocaine or phenytoin for ventricular arrhythmias & amiodarone for AF)
- Potassium (if hypokalemic)
- Digitalis antibody(DIGIBIND): the Fab fragment of the antibody binds to digoxin to form a complex that is excreted by the kidney.

## Other treatments

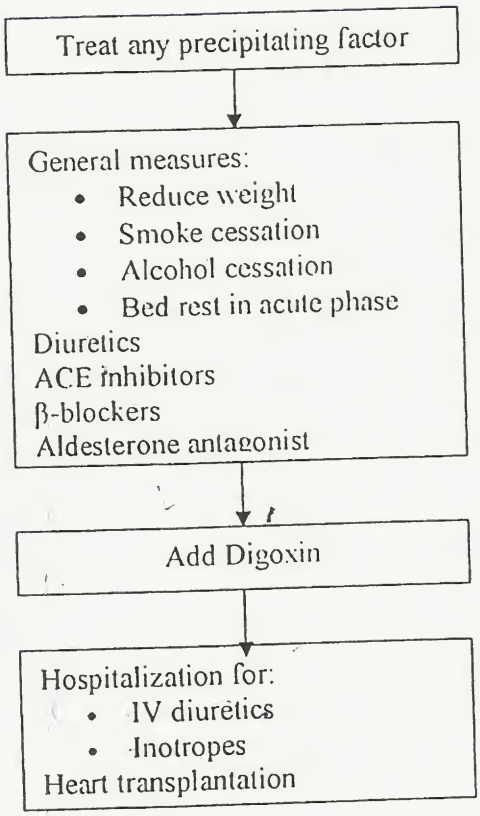
- **Vasodilators**
  Used in patient with severe heart failure.
  - Nitrates → venodilatation→ ↓preload
  - Hydralazine→ arterial dilators→ ↓reduce afterload

- **ICD** (Implantable Automatic Defibrillator) is used if the pt is having many ventricular extrasystoles on Holter ECG.
  ICD are shown to improve the prognosis

- **Cardiac Resynchronization** is used in patients with heart failure and LBBB
  Cardiac Resynchronization is shown to improve the prognosis

Left Bundle Branch Block






Drugs that **improve prognosis & reduce hospitalization** in heart failure:

- ACE inhibitors
- Spironolactone
- Beta-blockers
- Hydralazine/long-acting nitrates
- ICD (Implantable Automatic Defibrillator)
- Cardiac Resynchronization

| Drugs causing Gynecomastia: Digitalis & spironolactone | Drugs causing edema: |
|---|---|

## *Complications of heart failure*

1. *Arrhythmias* — Atrial fibrillation; ventricular arrhythmias (ventricular tachycardia, ventricular fibrillation); Bradyarrhythmias.
   - Ventricular fibrillation is responsible for one half of deaths in Heart failure
   - Treat arrhythmias only if symptomatic because ALL antiarrhythmic drugs are PROARRHYTHMIC
   - ICD (Implantable Automatic Defibrillator) is shown to improve prognosis in pt with severe heart failure by stopping ventricular arrhythmias
2. *Thromboembolism.* Venous (DVT & PE) due to ↓ mobility of the patient, or Arterial (stroke) due to arrhythmias.
3. *Gastrointestinal* — Hepatic congestion and hepatic dysfunction; malabsorption
4. *Musculoskeletal* — Muscle wasting
5. *Respiratory* — Pulmonary edema (Respiratory Failure type 1) ↓ Or
6. *Renal failure* due to ↓ cardiac output
7. *Electrolyte abnormalities* – Hypo or Hyperkalemia & Hyponatremia

## *Prognosis*

It carries a **very poor prognosis** with about 50% of cases will die within 2 years. And the cause of death is usually either ventricular arrhythmia or MI.



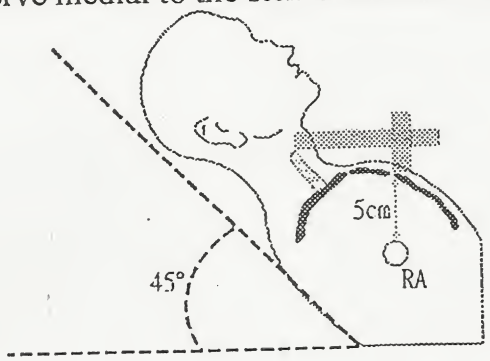
Poor prognostic factors for heart failure:

1. Male + Old + Sever symptom.
2. ↑BNP concentration + Impaired renal function [↑BUN].
3. Coronary artery disease
4. ↓ Ejection fraction ↓ & Hypotension
5. Hypokalemia & Hyponatremia

# Examination of the CVS

## *Examination of the JVP*

The patient should be in **45°** & the **height** and **waves** of the JVP should be assessed. The head should be supported on a pillow and turned slightly to left to see the right internal jugular vein. Observe medial to the sternomastoid muscle.




Q. What are the difference between JVP and Carotid pulse by examination?

| JVP | Carotid pulse |
|---|---|
| More visible than palpable | More palpable than visible |
| Obstructed by finger pressure | Not obstructed |
| ↓ with inspiration | No |
| Tow waves per cardiac cycle | Only one per cardiac cycle |
| More prominent when put pressure on abdomen [hepatojugular reflex] | No effect by Abdominal pressure |
| Varies with posture | No |
| When very high it moves ear lobe | Does not move ear lobe |

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### Assessment of the height

It is normally < 4 cm. This is equal to right atrial pressure of 9 cm of water as in this position, the manubriosternal angle is about 5 cm above the centre of the right atrium.

**Raised JVP (> 4 cm)** causes are:

1- Right-sided heart failure.
2- Tricuspid or Pulmonic valve disease.
3- Constrictive pericarditis.
4- Pericardial tamponade.
5- Obstruction of superior vena cava.
6- Fluid overload e.g. renal disease.

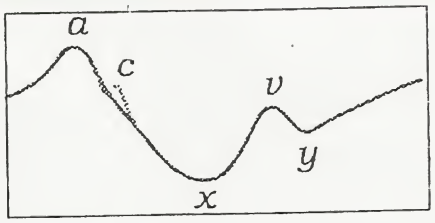
## Waveforms of the JVP

**Waves**

- *a* - presystolic; produced by right atrial contraction
- *v* - occurs in systole; due to increased blood in right atrium from venous return

**Descents**

- *x* – due to atrial relaxation
- *y* - tricuspid valve opens and blood flows in to the right ventricle




**Abnormalities of the JVP**

- Absent a wave → atrial fibrillation
- Dominant a wave
  1. Pulmonary stenosis
  2. Pulmonary hypertension
  3. Tricuspid stenosis
- **Cannon a wave**
  1. Complete heart block IV.
  2. Ventricular tachycardia [VF] → Cardiac Arrest.
- Dominant v wave →Tricuspid regurgitation
- Absent x descent→ Atrial fibrillation
- ↑ JVP and no wave forms → Superior Vena cava obstruction

JVP normally falls with inspiration but may rise (**Kussmaul's sign**) in constrictive pericarditis. [NOT pericardial tamponade]

VIM

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## (2) *Examination of the pulse*

1. **Rate**: normal: 60-100 beat/min. if<60 its bradycardia, and if >100 its tachycardia
2. **Rhythm**
   - Regular → Normal
   - Regularly irregular → Bigeminy [1 normal 1 VEB] (Digitalis toxicity)
   - Regularly irregular → Trigeminy [2 normal 1 VEB] (Digitalis toxicity)
   - Irregularly irregular → **Atrial fibrillation**
3. **Volume**
   - Small → Mitral stenosis, Aortic Stenosis, Heart failure
   - Good → Normal
   - Large → Same causes as collapsing pulse
4. **Condition of the wall**: Good (normal) or Hard (atherosclerosis)
5. **Special characters**:
   - *Slow rising pulse* : Felt by thumb over the carotid artery and it is characterized by delayed upstroke. Cause: **Aortic stenosis**.
   - *Bounding = collapsing pulse*: Felt with the thenar eminence over the radial artery. It occurs when diastolic is <60 mmHg & PP > 60 mmHg Causes: • **Aortic regurgitation** • **Hyperdynamic circulation** [Fever, Anemia, ↑T4, Pregnancy].
   - *Pulsus bisferiens*: Felt by thumb over the carotid artery and it is characterized by double systolic pulsation. Cause: **mixed AR & AS**.
   - *Pulsus alternans*: felt at the radial artery and is characterized by Regular alteration in volume amplitude (**severe LVF**).
   - *Pulsus paradoxus*: systolic BP falls >10 mmHg in during inspiration. Causes: **Pericardial tamponade. Severe asthma**.
   - *Jerky pulse*: hypertrophic obstructive cardiomyopathy, MR.
6. **Synchronicity:**
   - Radio-Radially [Delay occurs in Subclavian art. stenosis]
   - Radio-Femorally [Delay occurs in Coarctation of aorta]

85

# Precordium Examination Checklist

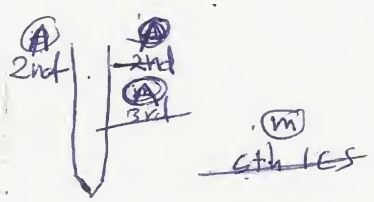
| Action | YES | NO |
|---|---|---|
| Take permission | | |
| Stand on the Right side of bed | | |
| Put pt in 45° [semi-recumbent] | | |
| Adequate Exposure | | |
| **Inspection** | | |
| 5 S | | |
| Deformity | | |
| Visible pulsation | | |
| **Palpation** | | |
| Apical beat position | | |
| Heave in Lt parasternal area | | |
| Thrill in all valve areas | | |

| Action | YES | NO |
|---|---|---|
| **Auscultation** | | |
| Warm the stethoscope | | |
| Timing by palpating Rt carotid | | |
| With diaphragm on 4 area | | |
| Bell on mitral & tricuspid area | | |
| Bell on mitral in Lt Lat position [for MS] | | |
| Ask the patient to Sit | | |
| Diaphragm on Lt 3rd ICS at expiration [for AR] | | |
| **I would like to check for:** | | |
| BP, JVP, Peripheral pulses, LLE. | | |
| **Ethics** | | |
| Dress the patient back | | |
| Thank the patient | | |

In case that you heard a murmur:

- Before sitting the pt:
  - Locate the site at which murmur is best heard
  - Listen for change with Inspiration [Rt side murmur ↑ but Lt side murmur ↓]
  - Listen for radiation in axilla [in MR]
  - Palpate the liver edge [in TR]

- After sitting the pt:
  - Listen for radiation in carotid [in AS]
  - Auscultate lung bases for crepitations of heart failure



# Precordium Examination

## ALWAYS put the pt in 45°

Adequate exposure is all chest and abdomen above the umbilicus

## Inspection

- Go to the end of the bed and look for deformities:[ a bulge over the precordium indicate that the disease is since childhood when cartilage was soft]
- Scars: Midline = Sternotomy. Lateral to midline called thoracotomy.
- Look for **Pulsations**:
  - Normally visible over the apex in thin people
  - Left parasternal pulsation → Right ventricular hypertrophy
  - Lt 2nd ICS pulsation → enlarged pulmonary artery
  - Rt 2nd ICS pulsation → aortic aneurysm
  - Diffuse apical pulsation → cardiomyopathy or post MI
  - Epigastric pulsation → 1- Normal in thin person, 2- Rt vent. enlargement, 3- Abdominal aneurysm, 4- Pancreatic cyst, 5- Pulsatile liver in TR

## Palpation

- Apical beat description:

i. **Apical beat definition**: most downward & most lateral palpable beat.

ii. **Site:**

a. Normally present in the 4th or 5th ICS at Midclavicular line
b. LV enlargement → Downward and laterally [in MR,AR]
c. RV enlargement → Only laterally

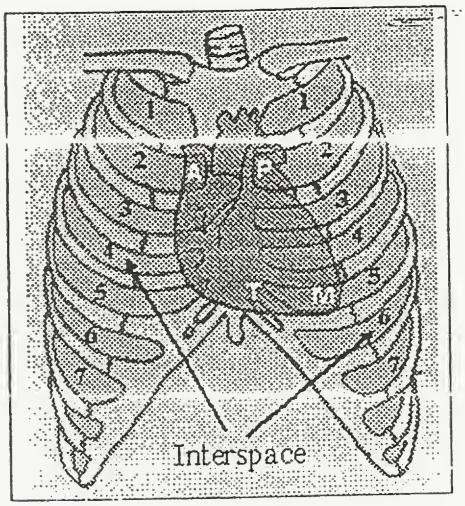
2- **Character:**

1. Normally as gentle non-sustained
2. Sustained = thrusting indicates ↑ load over left ventricle e.g. AS, AR, MR.
3. Tapping in MS [due to palpable S1]
4. Diffuse in cardiomyopathy or post MI
5. Double apex beat in Hypertrophic cardiomyopathy
6. Impalpable beat occurs in:
   - Obesity
   - Heart under the rib
   - Hyperinflated chest
   - Pericardial effusion
   - Dextrocardia

- Palpation for Lt parasternal heave which indicates Rt ventricular hypertrophy
- Palpation for Thrill in all valve areas. [Thrill is a palpable murmur]

*Location of the valve area*

**Aortic**: 2nd R ICS at right sternal border
**Pulmonic**: 2nd L ICS at left sternal border
**Second Aortic**: 3rd L ICS at left sternal border
**Tricuspid**: 4th L ICS at lower left sternal border
**Mitral** (apical): 5th L ICS at MCL




## Auscultation

Comment on S1 + S2+ added sound + Murmur

ALL heart sound are High-pitched = Heard with Diaphragm **Except**: Mid-diastolic murmur of MS, S3 & S4 which are Low-pitched and heard with Bell.
Uses of the bell: to listen for Mid-diastolic murmur of MS, S3 & S4

*S1*

- Heard with diaphragm, loudest at apex
- Due to closure of the mitral valve
- *Loud* S1: Mitral stenosis.
- *Soft* S1: Mitral regurgitation.

S2

- *S2* heard at Lt & Rt 2nd ICS with the diaphragm
- Its due to closure of Aortic & Pulmonic valves so it has 2 components
- *Loud* A2: Systemic hypertension.
- *Soft* A2: Aortic stenosis. Aortic regurgitation.
- *Loud* P2: Pulmonary hypertension.
- *Soft* P2: Pulmonic stenosis .

| *Added sounds* | |
|---|---|
| *S3* | • Low-pitched, heard best with bell at apex, it follows S2.<br>• Caused by rapid ventricular filling<br>• Normal in children, pregnant and adults < 35 years.<br>• After age 35, S3 indicates LVF or volume overload as in MR or AR. |
| *S4* | • Low-pitched, heard best with bell at apex, it precedes S1.<br>• Caused by atrial contraction into a noncompliant ventricle.<br>• Found in Heart failure, Aortic stenosis, Hypertension, IHD. |
| *Opening Snap* | By diaphragm High-pitched; follows S2, heard at apex in mitral stenosis (MS); when absent it indicates sever disease. |
| *Pericardial rub* in pericarditis | |
| *Metallic click of prostatic valves* | |

# Heart Murmurs

Murmurs are **not** added sound

| DDx of murmur |
|---|
| SYSTOLIC MURMURS<br>Ejection-type : [crescendo-decrescendo murmur]: begins after S1<br>• Aortic valve stenosis<br>• Hypertrophic obstructive cardiomyopathy<br>• Aortic & Pulmonic flow murmur [anemia]<br>• Pulmonic valve stenosis<br>Pansystolic: begins with S1<br>• Mitral regurgitation<br>• Tricuspid regurgitation<br>• Ventricular septal defect<br>Late-systolic:<br>• Mitral or tricuspid valve prolapse |
| DIASTOLIC MURMURS<br>• Early diastolic Aortic valve regurgitation<br>• Mid diastolic Mitral stenosis or Tricuspid stenosis<br>• Pulmonic valve regurgitation |
| CONTIOUS MURMURS<br>➢ Patent ductus arteriosus |



Comment on the 5 characteristics of murmurs:

1. Site of murmur [See above]
2. Timing [systolic or diastolic]
3. Grade = Intensity
4. Radiation (axilla for MR carotid for AS)
5. Variation with respiration.

During Inspiration there is ↑ -ve pressure in chest which → ↑ in venous return to the right ventricle from inferior vena cava→ Stretching of Rt vent → more powerful contraction → Murmurs in Rt side are louder during inspiration.
During Inspiration there is ↑ -ve pressure in chest which → ↓ venous return to the left ventricle form the lungs → ↓ Stretching of Rt vent → less powerful contraction → Murmurs in Lt side are Quiter during inspiration.
On Expiration the opposite occurs [Rt side are weaker & Lt side are louder]

Intensity of murmur

Grade I: Just audible in a quite room, with patient's breath held, never immediately
Grade II: Quite
Grade III: Moderately loud
**Grade IV**: Loud and accompanied by **thrill on palpation**.
Grade V: very loud with only the rim of the diaphragm is need to hear the murmur
Grade VI: heard with stethoscope over the chest

# Investigations in the CVS

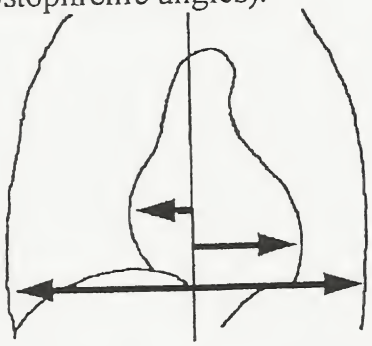
For any CVS case:
1- ECG
2- CXR
3- Echo

## Chest-X-ray

- The heart size:

Comment on the heart size **only** with PA chest-x-ray.

**Cardiothoracic ratio**: This is the transverse cardiac diameter [the horizontal distance between the most rightward and leftward borders of the heart seen on a postero-anterior (PA) chest radiograph] divided by the transverse chest diameter (measured from the inside rib margin at the widest point above the costophrenic angles).



**Cardiothoracic ratio should be < 0.5 = 50%.**

Causes of a ratio > 50% include:

1- Dilatation of cardiac ventricles.
2- Hypertrophy of cardiac ventricles
3- Pericardial effusion

Dilatation of each of cardiac chambers has characteristic appearance on:

- **Left atrial dilatation**:
  - **Mitralization** of the left heart border [Straightening of left heart border due to prominence of the Lt At appendage].
  - Widening = splaying of the angle of the carina.
  - Double cardiac shadow to the right of the sternum
- **Right atrial** enlargement projects from the right heart border
- **Left ventricular dilatation**:
  - ↑ Cardiac silhouette, with rounding of the left heart border.
  - Prominence of the left lower heart border.
- **Right ventricular dilatation**:
  - ↑ Cardiac silhouette with apex displaced upward & Straightening of the left heart border

## Echocardiography

There are 2 types of echo Transthoracic & Transesophageal

Indications of echocardiography:

1. Valvular disease
2. Ventricular performance
3. Cardiac source of embolism [Transesophageal is better]
4. Endocarditis [Transesophageal is better]

## CT & MRI

CT is most useful for imaging the aorta in suspected aortic dissection.

MRI is used to assess ventricular wall motion



Computarized tomography

magnatig resonat Imaging

# ECG

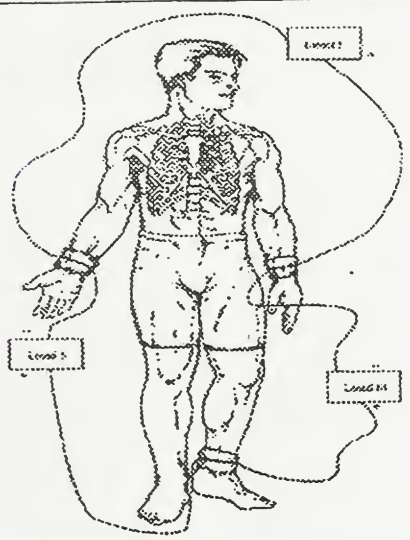
Q. What is ECG?!
ECG [ElectroCardioGram] is a drawing showing the electrical activity of the heart.
(Electro- from electric, Cardio- means heart, & -Gram means a drawing)

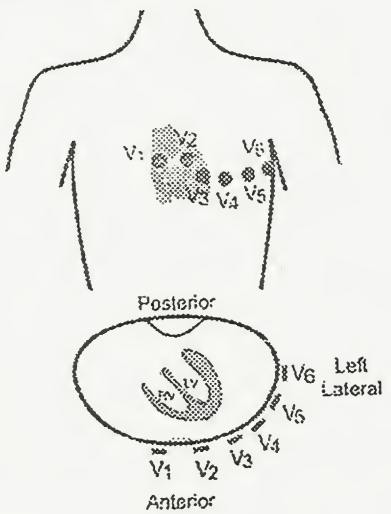
Q. What generates the electrical activity of the heart?
The depolarization & repolarization of cardiac tissues (muscle & conducting system)

When the electrical activity of the heart reaches the body surface it's measured by electrode of the ECG machine positioned on the skin.

*The leads*
- **6 Frontal (limb)** leads
  - Bipolar leads (I,II, & III)
  - Augmented limb leads (aVL, aVR,& aVF)]
- **6 Horizontal ( precordial)** leads (V1-V6)

| Lead Type | Positive Input | Negative Input | |
|---|---|---|---|
| **Standard Limb Leads** *(Bipolar leads)* | | | |
| I | Left arm | Right arm | |
| II | Left leg | Right arm | |
| III | Left leg | Left arm | |
| **Augmented Limb Leads** *(Unipolar leads)* | | | |
| aVR | Right arm | Left arm + left leg | |
| aVL | Left arm | Right arm + left leg | |
| aVF | Left leg | Left arm +left arm | |
| **Precordial Leads** *(Unipolar leads)* | | | |
| $V_1$ | Right sternal margin at 4th intercostal space | | Wilson central terminal |
| $V_2$ | Left sternal margin at 4th intercostal space | | |
| $V_3$ | Midway between $V_2$ and $V_4$ | | |
| $V_4$ | Left midclavicular line at 5th intercostal space | | |
| $V_5$ | Left anterior axillary line at 5th intercostal space | | |
| $V_6$ | Left midaxillary line at 5th intercostal space | | |








## Q. How ECG is drawn ?

The shape of the ECG depends on:

1. Speed and calibration of the ECG machine.

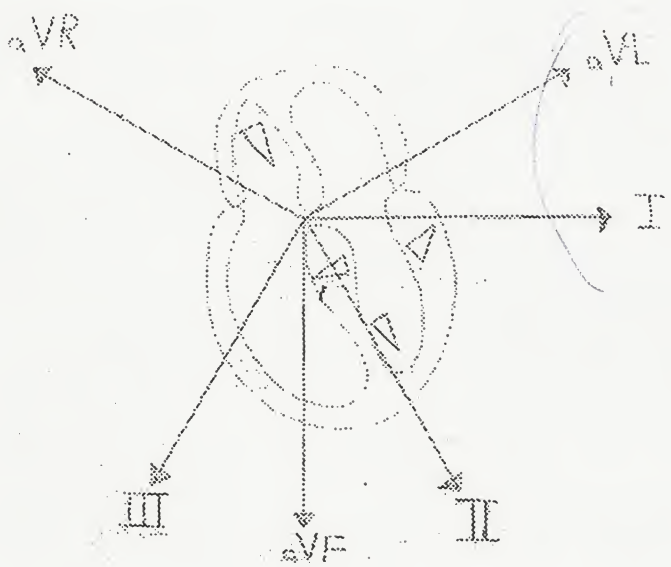
By convention the calibration is: 10 mm = 1 mV & Paper speed: 25 mm per second

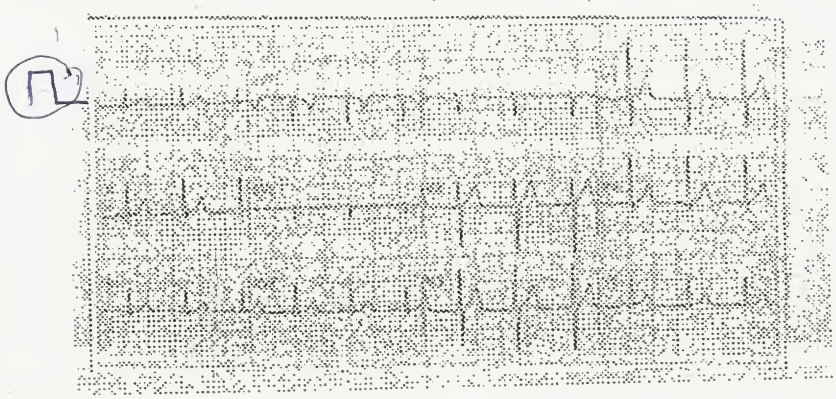
This will result in: • Each small (1 mm) square = 0.04 s
• Each large (5 mm) square = 0.2 s

2. The Relationship between Lead vectors and Cardiac vectors which depends on:
   a. The direction of the depolarisation:
      i. Depolarisation towards electrode → positive deflection
      ii. Depolarisation away from electrode → negative deflection
   b. The angle between the lead and cardiac vectors:
      i. Perpendicular angle will result in isoelectric small wave
      ii. Vectors with 0 or 180 will result in a large amplitude

3. By convention the X-axis in the ECG is the time and the Y-axis is the amplitude which depends on the magnitude of the wave:
   - **More powerful contraction → higher amplitude**
   - **Longer time in transmission → longer interval**





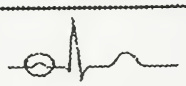
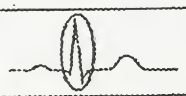
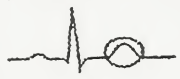
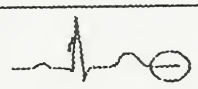



The ECG is composed of 3 waves and 2 segments:

- **P wave:** represents atrial depolarization
- **PR segment:** represents the delay at the AV node
- **QRS wave:** represents ventricular depolarization
- **ST segment:** represents time at which the ventricles remain depolarized
- **T wave**: represents ventricular repolarization

[Note: atrial repolarization occurs at the same time of ventricular depolarization which is of greater electrical power and masks the atrial repolarization]

[Note: **PR segment** extends from the end of P wave until the beginning of the QRS complex while **PR interval** extends from the beginning of the P wave until the beginning of the QRS complex]

| Electrical Activity | Graphic Depiction | Associated Pattern |
|---|---|---|
| Atrial Depolarization |  | P Wave |
| Delay at AV Node |  | PR Segment |
| Ventricular Depolarization |  | QRS Complex |
| Ventricular Repolarization |  | T Wave |
| No electrical activity |  | Isoelectric Line |

atrial contraction

ventricular contraction

ventricular relaxation

QRS drawing:

- If the first deflection is downward it is called Q wave.
- If the first deflection is upward it is called R wave either it was preceded by Q wave or not.
- The any downward deflection after the R wave is called S wave.
- An upward deflection after the S wave is called **R`**.

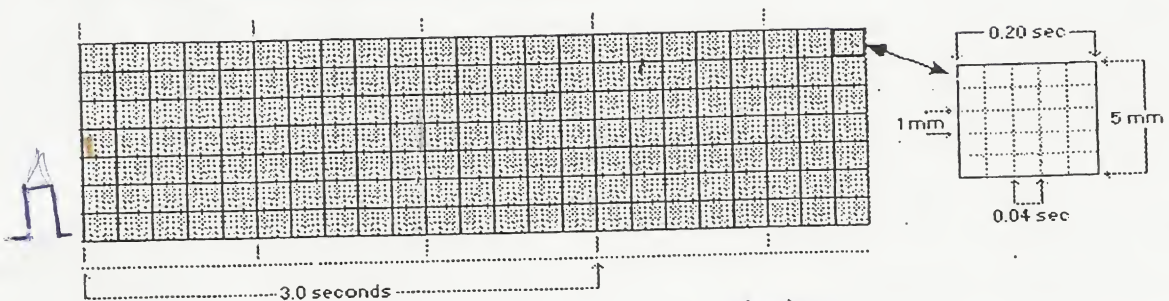
# ECG Interpretation

## Steps of ECG interpretation

1. Name & Date
2. Speed & Calibration
3. Rhythm "Sinus or not"
4. Rate
5. Axis
6. Waves and Intervals:
   - P wave
   - PR interval
   - QRS
   - ST segment
   - T wave

## Speed and Calibration:

- Time is on the horizontal axis while Voltage is on vertical axis
- Usual speed is 25mm/s:
  - ECG paper has small & large squares
  - Small square = 1mm
  - Each large square contains 5 small squares = 5 mm
  - 1 large square = 0.2 sec
  - 1 small square = 0.04 sec
  - 1 minute has 300 large squares or 1500 small squares
- Calibration is usually 1mv= 10 mm




## Rhythm

The aim is to tell if the rhythm is sinus or not. [Sinus rhythm means that the electrical activity of the heart starts in the SA node]

Characteristics of sinus rhythm

1. Each P-wave is followed by QRS
2. P waves in leads I & II is upward
3. P-R interval is regular
4. R- R interval is regular

## Rate

When regular → 300/R-R interval

When irregular → count the pulses in 15 large squares and multiplies by 20

## Axis

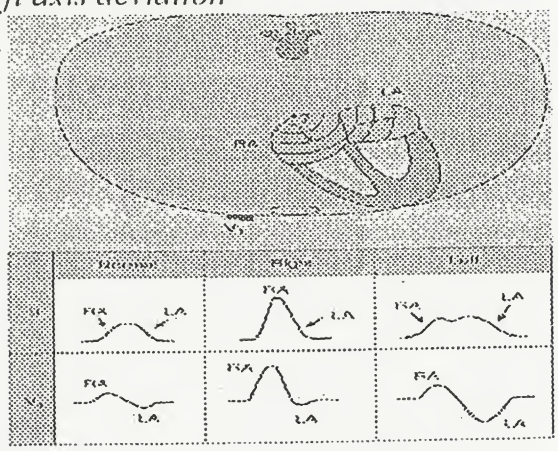
- Normal axis is between -30 to +110
- Right axis deviation is axis > + 110
- Left axis deviation is axis < - 30

| | | | |
|---|---|---|---|
| Lead I | +ve | -ve | +ve |
| Lead III | +ve | +ve | -ve |
| | Normal | Right axis | Check Lead II |

| | |
|---|---|
| +ve | -ve |
| Normal | Left axis |

| Causes of right axis deviation(+110to +150) | Causes of left axis deviation(-30 to -90) |
|---|---|
| Normal in young children and tall adults | Obesity |
| Left posterior hemiblock | Left anterior hemiblock |
| RVH & RBBB | LBBB |
| Anterolateral MI | Inferior MI |
| Pulmonary embolism | Emphysema |
| | Hyperkalemia |

*Note: not all left ventricular hypertrophy cause left axis deviation*



## Waves and Intervals

### P-wave

Look for the P wave in leads **II** & **V1**
Normal P wave characteristics:

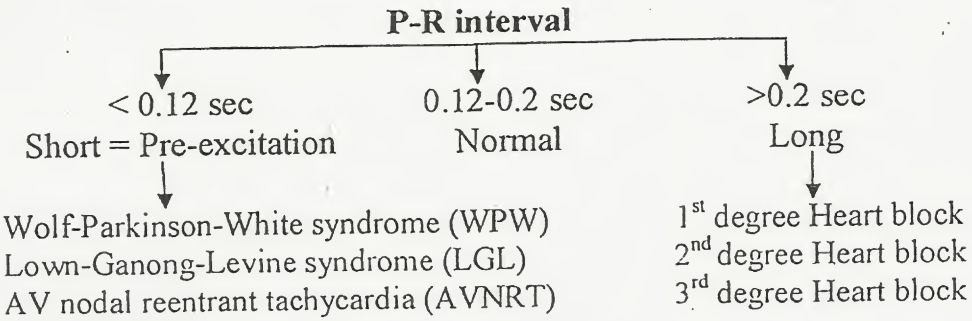
1 Width < 0.12 sec
2 Amplitude < 2.5 mm
3 –ve only in aVR

*Abnormalities*:

1. Width > 0.12 sec & notched is called **P-Mitral** and indicates Left Atrial dilation as in MS or ↑ Left Atrial filling pressure as in LVF or MR.
2. Amplitude > 2.5 mm = it's called **P-Pulmonal** which indicates Right Atrial dilatation as in TS or ↑ Right Atrial filling pressure as in RVF or TR or PE.
3. -ve P wave at lead I, II, III & +ve in aVR → Junctional Rhythm
4. P wave not looking like other P waves → Atrial ectopic
5. Multiple P wave with Saw-Toothed → Atrial flutter
6. Absent P wave or replaced by F waves→Atrial Fibrillation

## P-R interval

It's the interval from the **beginning** of P wave to the beginning of the QRS



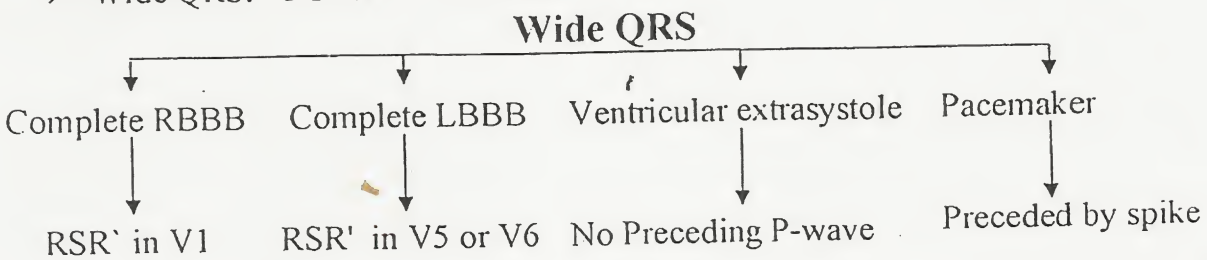

## QRS wave

Normal QRS:

1. Width is < 3 small squares (0.12sec)
2. Normal Q wave: <1 small square & < 1/3 of R wave height.
3. Amplitude:
   - R-wave in V1 & V2 < 5mm
   - R-wave at V5 & V6 < 25 mm
   - S-wave in V1 or V2+ R-wave of V5 or V6 < 35 mm

Abnormal QRS:

- Wide QRS: >3 small = > 0.12 sec




| Difference between Atrial & Ventricular arrhythmias | | |
|---|---|---|
| | Atrial | Ventricular |
| QRS | QRS look like the one before it | QRS doesn't look like the one before it |
| | P wave | No P wave |
| | Narrow QRS | Wide QRS |
| | Normal T wave | T wave inversion |
| | Not present | Capture beat and fusion beats |
| Response to carotid message | Yes | No |

- Abnormal Q-wave = > 1 small square & > 1/3 of the R-wave. It occurs in MI.
- Ventricular Hypertrophy:



1. Right ventricular overload:
   1. R:S > 1
   2. R-wave in V1 or V2 > 5mm
2. Left ventricular overload:
   1. R-wave at V5 or V6 ≥ 25 mm
   2. S-wave in V1 or V2 + R-wave of V5 or V6 ≥ 35 mm

| Vent. Enlargement | Causes of RVH | Causes of LVH |
|---|---|---|
| | PS<br>PHT<br>HCM | AS<br>HT<br>HCM |
| | Causes of RV dilatation | Causes of LV dilation |
| | TR<br>PR<br>Dilated CM | AR<br>MR<br>Dilated CM |

## ST segment

Normal ST segment:
1. Isoelectric
2. Up to 1mm up or down in the limb leads
3. Up to 2 mm up or down in Precordial leads

If UP > 1mm in limb leads OR >2mm in precordial leads

*ST elevation*:
1. Acute ST-elevation MI (STEMI).
2. Prinzmetal's angina (coronary spasm)
3. Pericarditis.
4. LV aneurysm.
5. LBBB

If DOWN > 1mm in limb leads OR >2mm in precordial leads + The depression is Horizontal or down sloping [not up sloping = normal]

*ST depression* :
1. Non-ST elevation MI (NSTEMI).
2. Ischemia (Stable & Unstable angina)
3. Digitalis effect
4. Strain (vent. hypertrophy → ischemia)

**Strain pattern**: ventricle hypertrophy → **Relative ischemia** and characterized by:
1- T-wave inversion
2- Asymmetric T-wave
3- ST depression

## T wave

Normal T wave:

1. Up to 1mm up or down in the limb leads
2. Up to 2 mm up or down in Precordial leads
3. Normal T wave is upward but may be normally inverted in lead III, V1, & V2.

T wave abnormalities:

- *Tall= Peaked T wave*
  - Hyperkalemia
  - Acute STEMI
- *Inverted T or Low T wave*:
  - Ischemia [Angina or MI]
  - Ventricular Strain pattern
  - Myocarditis
  - Digitalis effect
  - Subarachnoid Hemorrhage.
  - Electrolyte abnormality:
    1. Hypokalemia.
    2. Hypocalcemia.
    3. Hypomagnisemia

**U wave**: a wave after T-wave occurs in **Hypokalemia**

---

**Q-T interval**

The QT interval includes both ventricular depolarization and repolarization.

- QT ↓ as the heart rate ↑.
- Q-T is measure from the beginning of QRS till the end of T wave.
- Q-T is corrected according to the rate: $\mathbf{QT_c} = QT/\sqrt{R\text{-}R \text{ interval}}$
- Normal QTc is < 0.44 sec

**Prolonged Q-T → Torsade de point**

| **Drugs:** | **Electrolyte:** | **Congenital syndrome:** |
|---|---|---|
| 1. Antiarrhythmics[Ia, Ic, III] | 1. Hypokalemia. | • Romano-Ward (AD) |
| 2. TCA | 2. Hypocalcemia | • Jervell and Lange-Nielson (AR + deafness) |
| 3. Erythromycin | 3. Hypomagnisemia | |

---

**For ECG change in MI see MI sheet**

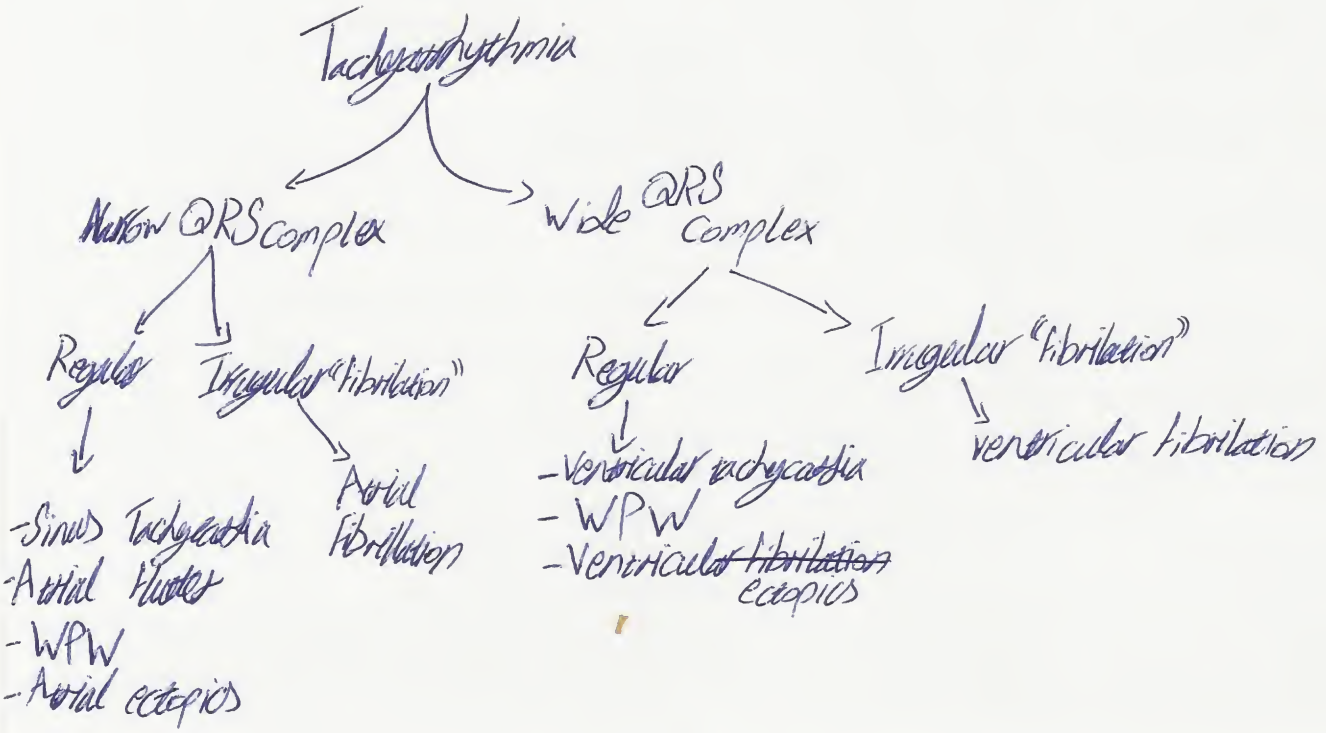
**For ECG changes in Myocarditis see Myocarditis sheet**

**For ECG changes in Pericarditis see Pericarditis sheet**

# ECG Abnormalities criteria

| Abnormality | Criteria |
|---|---|
| Asinus rhythm | P-wave is not followed by QRS |
| Abnormal rate | HR < 60 its bradycardia or HR > 100 in tachycardia |
| Right axis deviation | Negative QRS in lead I |
| Left axis deviation | Negative QRS in lead II **AND** lead III |
| Rt atrial enlargement | P wave in II >2.5 mm in amplitude |
| Lt atrial enlargement | P wave duration in lead II ≥ 0.12 sec |
| AV block | PR interval > 0.20 sec |
| Pre-excitation | PR interval < 0.12 sec |
| LVH | R wave in V5 or V6 > 35 mm **OR**<br>S wave in V1 or V2 + the R wave in V5 or V6 >35mm |
| RVH | R wave in V1 >5mm |
| ST segment elevation | ST segment >1 mV in limb leads OR<br>ST segment >2 mV in precordial leads |
| Pathologic Q wave | Duration ≥ 0.04 sec **AND**<br>Amplitude> 1/3 the height of the R wave |
| Peaked T wave | T wave amplitude >6 mm in limb leads **OR**<br>T wave amplitude> 10 mm in precordial leads |
| LBBB | QRS duration > 0.12 sec **AND** RSR` pattern in leads I, V5, V 6 |
| RBBB | QRS duration > 0.12 sec **AND** RSR` pattern in V1, V2 |

| Heart wall MI | ECG changes | Coronary artery |
|---|---|---|
| **Anteroseptal** | V2-V4 | Left anterior descending |
| **Lateral** | I, aVL +/- V5-6 | Left circumflex |
| **Anterolateral** | V2-6, I, aVL | Left main stem |
| **Inferior** | II, III, aVF | Right coronary |

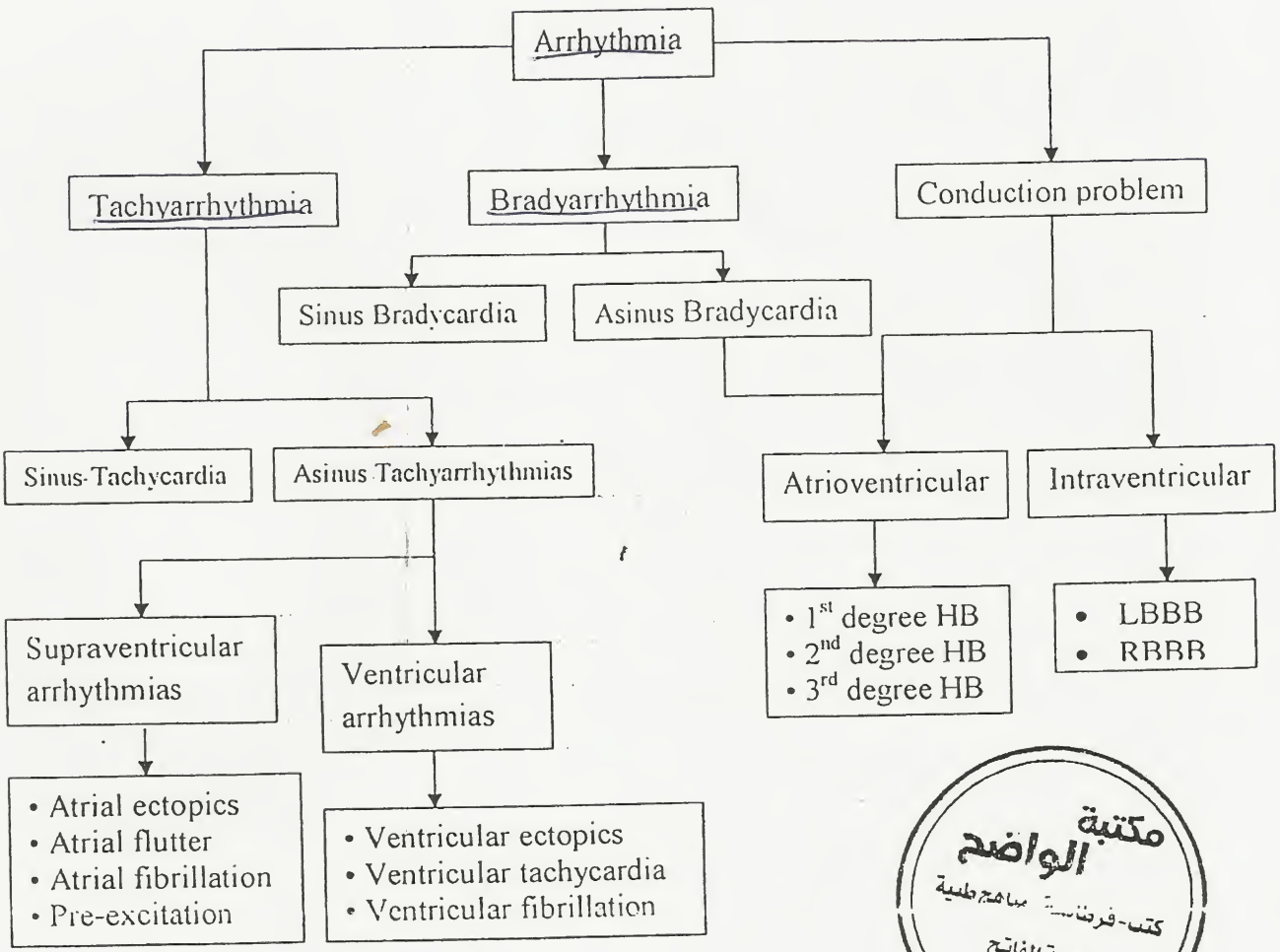

Tachyarrhythmia
Narrow QRS complex
Wide QRS complex
Regular
Irregular "fibrilation"
Regular
Irregular "fibrilation"
-Sinus Tachycardia
-Atrial flutter
-WPW
-Atrial ectopics
Atrial fibrillation
-Ventricular tachycardia
-WPW
-Ventricular ectopics
ventricular fibrilation

# ARRHYTHMIAS

*Definition of Arrhythmia*: disorders of Heart rate, Rhythm, & conduction.

Normal Heart rate is between 60-100 b/min, Tachycardia: is HR of >100 b/min, while Bradycardia: is HR < 60 b/min.

The rhythm is classified to either Sinus (i.e. originating from the SA node) or Asinus which mean any rhythm other than the SA node.



# Bradyarrhythmias

***Definition:*** HR < 60 b/min with Sinus or Asinus rhythm.

***Symptoms*** of ALL Bradyarrhythmias:

- Asymptomatic
- Dizziness & Syncope
- Confusion
- CHF

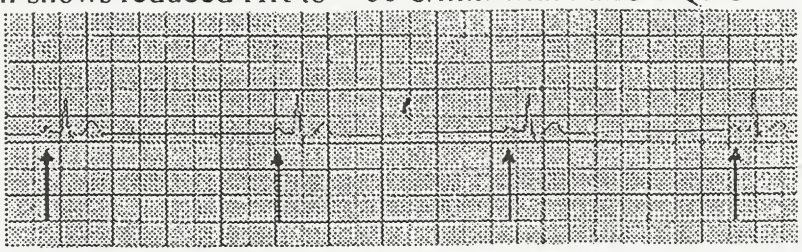
## Sinus Bradycardia

***Definition:*** HR < 60 b/min due to ↓ firing of the SA node

*Causes:* Idiopathic degeneration (sick sinus syndrome) & drugs are most common.

| Cardiac | Metabolic | Systemic | Idiopathic |
|---|---|---|---|
| 1- MI<br>2- Sick sinus syndrome<br>3- Acute HT | 1- Hypothyroidism<br>2- **Cholestatic jaundice**<br>3- Hypothermia<br>4- Drugs (β-blocker, digoxin, CCB) | 1- ↑ ICP due to any cause (Cushing Triad)<br>2- **Relative Bradycardia**<br>• Typhoid fever<br>• Brucellosis | 1- Sleep<br>2- Athletics |

**Dx:** ECG which shows reduced HR to < 60 b/min with narrow QRS



### *Sick sinus syndrome*

Combination of symptoms (dizziness, syncope, fatigue, confusion, & CHF) + SA node dysfunction due to degeneration or ischemia, its more common in **elderly**. The SA node dysfunction may be :

- Sinus **bradycardia** or Sinoatrial block (sinus arrest) **or**
- Alternating **bradycardia** with **tachycardia** (Atrial flutter or AF)

**Rx:**

- Asymptomatic no treatment.
- If Symptomatic atropine. If still symptomatic → Permanent Pacemaker.

# AV Block [1st, 2nd, & 3rd degree Heart block]

When the AV node is blocked the escape rhythm may arise from:

1. **Proximal His bundle** with **40- 60 beats/min** with narrow QRS, which is electrophysiologically & hemodynamically stable
2. **Distal His-Purkinje system** with **25-40 beats/min** with wide QRS. And its electrophysiologically unstable (i.e. can progress to complete heart block) & hemodynamically unstable (i.e. can cause symptoms of dizziness and syncope)



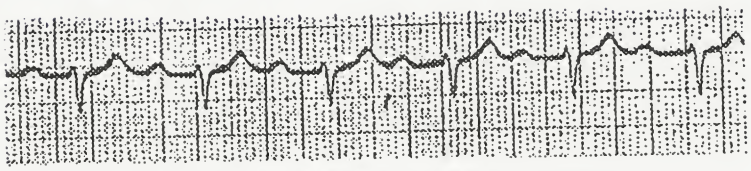
## Causes of AV block

1. Congenital
2. Increased vagal tone
3. **Drugs** (β-blockers, calcium channel blockers, **digitalis**)
4. **MI** → **Inferior MI** → ↑ **vagal tone** which is usually **transient**
   → **Anterior MI** → **due to ischemia**
5. Degenerative disease = idiopathic fibrosis in the AV junction.

## **First-degree AV block** (prolonged AV conduction)

**ECG:** Prolonged, constant PR interval >0.20 s & every atrial beat is conducted to the ventricle. QRS is narrow.

It's only a delay in AV node (i.e. no escape rhythm) → Pt rarely symptomatic



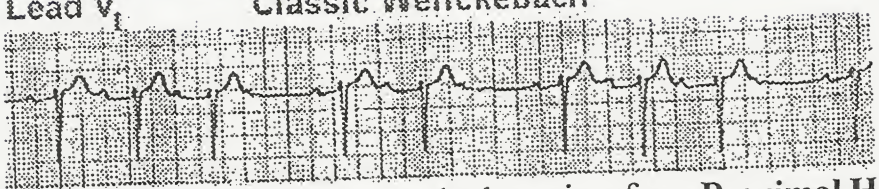
1st degree AV block (PR = 280 ms)

Cause: as above + **Rheumatic fever**

## **Second-degree AV block** (intermittent AV block)

(a) **Mobitz type I (Wenckebach) block**

**ECG:** There is a progressive prolongation in the P-R interval until a generated P wave is not conducted. Narrow QRS. There are usually 3-5 beats in a cycle.



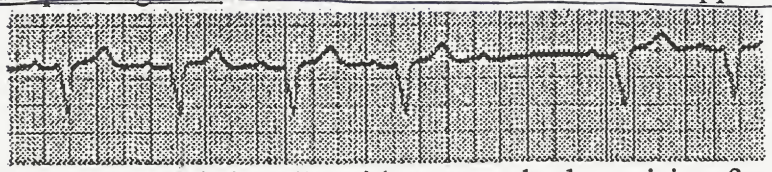

It's a disease of AV node only and escape rhythm arises from **Proximal His bundle** Which is electrophysiologically & hemodynamically stable → So Rx in only if the pt is symptomatic which is rare. [Rx: Atropine. If symp. still→ Permanent Pacemaker]

Causes: of 3rd degree heart block

- MI
- abscess formation "IE"
- Sarcoidosis
- Digoxin
- lime disease
- β blockers
- calcium channel blocker
- Trypanosoma cruzi

(b) **Mobitz type II block**

**ECG:** There is no prolongation of the P-R interval before the dropped beat.



It's a disease of AV node & His bundle with escape rhythm arising from **Distal His-Purkinje system** which is electrophysiologically unstable (i.e. can progress to complete heart block) & hemodynamically unstable (i.e. can cause symptoms of dizziness and syncopal attack) → so patient should be treated even if he is asymptomatic because of the fear from complete heart block.

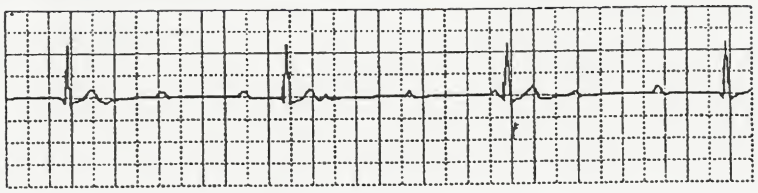
## Third degree/complete heart block

No impulses are conducted from the atria to ventricles.

ECG: show atrioventricular dissociation with wide QRS Fixed R R interval

**C/P: Severe symptomatic bradycardia** characterized by a heart rate of 25to 40 bpm due to escape rhythm arising from **Distal His-Purkinje system**

- JVP may show **canon a waves**
- **Adam-Stock syndrome** it is syncope due to intermittent complete heart block or transient asystole. Skin pallor may be followed by flushing. And if the anoxia lasts more than a few seconds a convulsion may occur.



## *Management of AV Block*

**AV block complicating acute myocardial infarction**

- First Degree & Mobitz I no treatment is indicated unless symptomatic → atropine
- Mobitz II & Complete heart block if in Acute Inferior MI → reliable escape rhythm + transient AV block due to ↑ vagal tone→ no treatment is required unless the patient is Symptomatic with atropine → if fails→ temporary pacemaker.
- Mobitz II & Complete heart block if in Acute Anterior MI → **un**reliable escape rhythm [may cause asystole] + the AV block is due to ischemia of the right and left bundles→ pacemaker should be inserted as soon as possible.

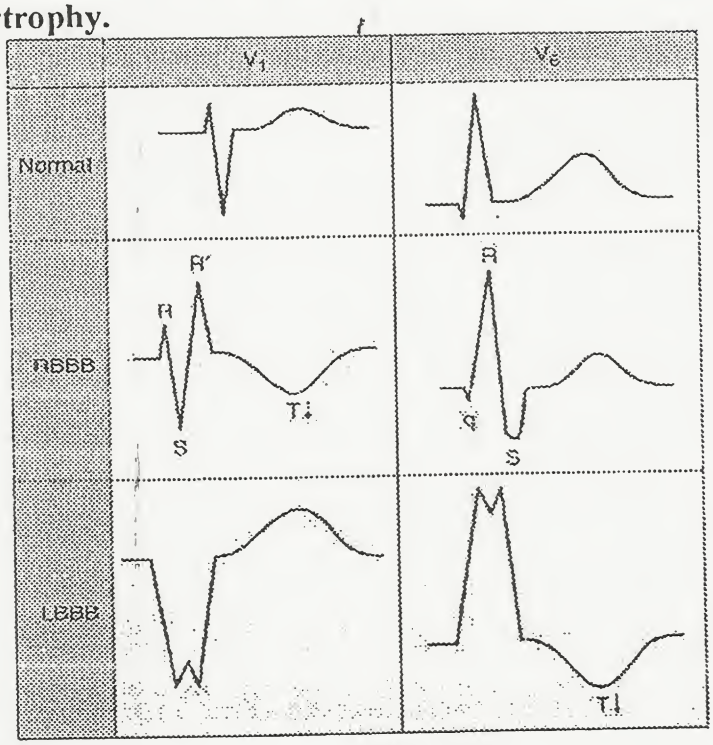
**Chronic AV block**

- First Degree & Mobitz I no treatment is indicated unless symptomatic → atropine
- Mobitz II & Complete heart block → **un**reliable escape rhythm [may cause asystole]→ permanent pacemaker is indicated except If the cause is congenital.

# Bundle Branch Block

| | RBBB [Right Bundle Branch Block] | LBBB [Left Bundle Branch Block] |
|---|---|---|
| **ECG criteria** | • QRS duration ≥ 120 msec<br>• Broad, notched R waves (RSR` patterns) in right precordial leads ($V_1$ and $V_2$) | • QRS duration ≥ 120 msec<br>• Broad, notched R waves (RSR`) also called M-shaped wave in lateral precordial leads ($V_5$ and $V_6$) ± leads I and aVL |
| Causes | 1. Normal variant<br>2. ASD<br>3. Pulmonary hypertension or PS<br>4. Pulmonary embolism<br>5. Ischemic heart diseases<br>6. Cardiomyopathies<br>7. Idiopathic fibrosis | 1. Hypertension or AS or MS<br>2. Ischemic heart diseases<br>3. Cardiomyopathies<br>4. Idiopathic fibrosis |
| Rx | • Treat the underlying cause if present<br>• In itself it does not require treatment | • LBBB is always an indication of heart disease.<br>• **Acute onset of LBBB is an indication for Thrombolytics.**<br>• If the pt is symptomatic or QRS is markedly prolonged → Pacemaker.<br>• In pt with HF & LBBB → Cardiac Resynchronisation is used. |

**In the presence of LBBB do not interoperate for Myocardial infarction or Left ventricular hypertrophy.**



# Tachyarrhythmias

***Definition:*** heart rate of >100 b/min with Sinus or Asinus rhythm.

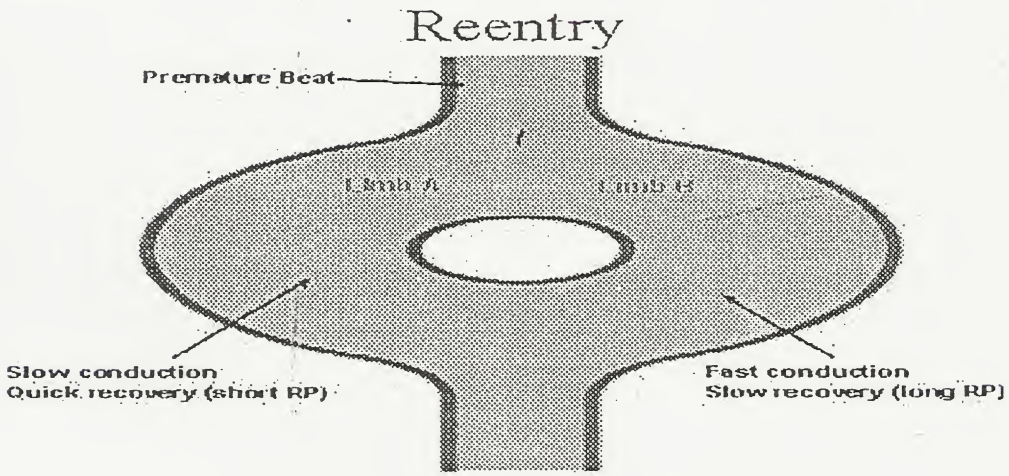
**All** tachyarrhythmias have the same causes:

| **Cardiac** | **Metabolic** | **Pulmonary** |
|---|---|---|
| 1- IHD | 1- Alcohol | 1- PE |
| 2- Hypertensive heart disease | 2- Thyrotoxicosis | 2- COPD |
| 3- Valve disease (esp MS) | 3- ↑ Sympathetic activity (Anxiety, Anemia) | **Idiopathic** |
| 4- Pericarditis | | |
| 5- Cardiomyopathy | 4- Drugs (β-agonists) | |
| 6- Heart failure | 5- Hypo- or hyperkalemia | |

## *Pathophysiology*

There are three main mechanisms:

- **Re-entry- Most common mechanism** its due to difference in conduction speeds and refractory periods.
- **Increased automaticity** results from repeated spontaneous depolarisation of an ectopic focus. (usually due to metabolic cause)
- **Triggered activity**: results form of secondary depolarisation arising from an incompletely repolarised cell membrane. e.g. ventricular arrhythmias in pts with ischemic heart disease.




## *Symptoms of all tachyarrhythmias:*

- Asymptomatic
- **Palpitation**
- Symptoms of heart failure (breathlessness)
- Dizziness & Syncope
- Polyuria [stretching of the atria during attack → release of ANP→ polyuria]

***Principles of management of tachyarrhythmias***

- In all tachyarrhythmias (except sinus tachyarrhythmia) if the pt is unstable perform DC shock.
- In supraventricular arrhythmias maneuvers that stimulate Vagus nerve may be used, and these maneuvers include:
  1. **Carotid artery massage**
  2. **Valsalva maneuver:** forcibly exhaling against a closed glottis (a closed airway)
  3. Immersion of pt face in ice

## Sinus Tachycardia

*Definition:* HR of > 100 b/min due to increased firing of the SA node
**ECG:** the only change is HR 100- 140 b/min
*Rx*: Treat the underlying cause & if symptomatic β-blockers

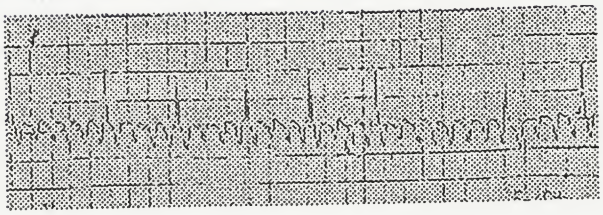
## Atrial ectopics

Atrial premature beats are usually preceded by a P wave of different shape. The following QRS complex is normal. Frequently the ECG demonstrates P waves that are inverted in the inferior leads (leads II, III, a VF) and upright in lead a VR, reflecting the origin of this arrhythmia from the inferior aspect of the atria.
Rx: No need

## Atrial Flutter

*Definition:* Atrial rate of 240 - <350 /min due to single large reentry circuit.
*Causes & Symptoms* → see above
*ECG:*

**Saw tooted P wave** atrial rate of 300/ min with 3:1 block the QRS complex is narrow



Rx: As Atrial Fibrillation + Catheter ablation is the treatment of choice

# Atrial Fibrillation VERY IMPORTAT

AF is the **most common** sustained arrhythmia. during the exam

***Definition:*** Spontaneous rapid atrial depolarization > 350/min due to multiple, reentry circuits around the atria.

AF→ ineffective atrial contraction → abnormal hemodynamic → thrombus formation if the AF duration is > 2 days.

*Causes*→ see above & Concentrate on Thyrotoxicosis & Mitral stenosis. & HTN

*Symptoms:* See above but here thromboembolic symptoms also occur:

- Thromboembolic symptoms (**CVA**, Renal infarction, Mesenteric infarction, **Limb ischemia**, MI).

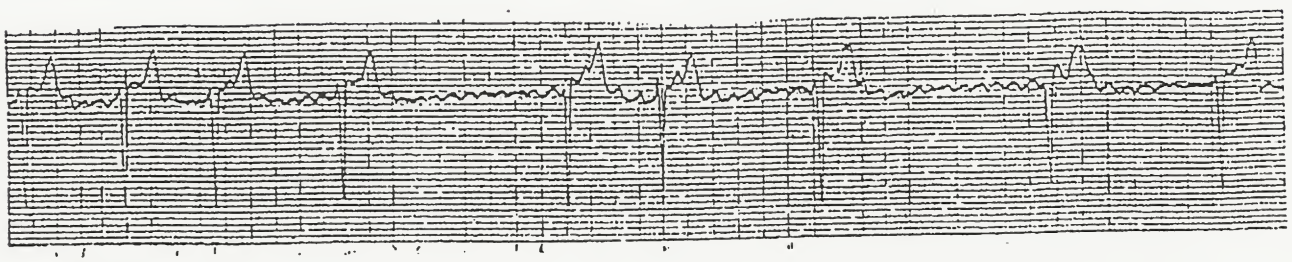
*Signs:* • Irregularly irregular pulse
- Absent a wave on the JVP (differentiates it from multiple vent ectopics)
- Pt may develop C/P of heart failure

-- absent P wave on the ECG

Classification of AF

- **Paroxysmal AF** Recurrent episodes ≥ 2 & episodes terminate spontaneously. β-blockers are the drug of choice in these cases.
- **Persistent AF** If AF is not self-terminating but can be terminated by cardioversion.
- **Permanent AF** there is continuous atrial fibrillation which cannot be cardioverted. So only rate control & prevention of thromboembolism are the treatment.

ECG: Asinus rhythm which is irregularly irregular, P wave is replaced by F wave, & QRS complex is narrow



## *Treatment*

- In acute AF precipitating factor should be looked for..
- If the patient becomes unstable electrical cardioversion is the Rx of choice "DC shock"
- Maneuvers that ↑ the vagal tone [Carotid artery message, Valsalva]

1- **Rate control:** ↓ Ventricular rate by ↓conduction through AV node using (Ideal rate 60-80 b\m):
   a. *Beta blockers should be used first followed by CCB & digoxin if rate uncontrolled.*
   b. *Calcium channel blockers* (verapamil or diltiazem).
   c. *Digoxin* if the pt has HF in addition to AF use digoxin first

2- **Conversion to sinus rhythm**: may be either electrical cardioversion or by drug.

a. If AF of less than 2 days DC (Direct Current) cardioversion under anesthesia with IV heparin.

b. If AF is of > 2 days but no atrial thrombus can be detected by esophageal echocardiography then again, DC cardioversion with IV heparin.

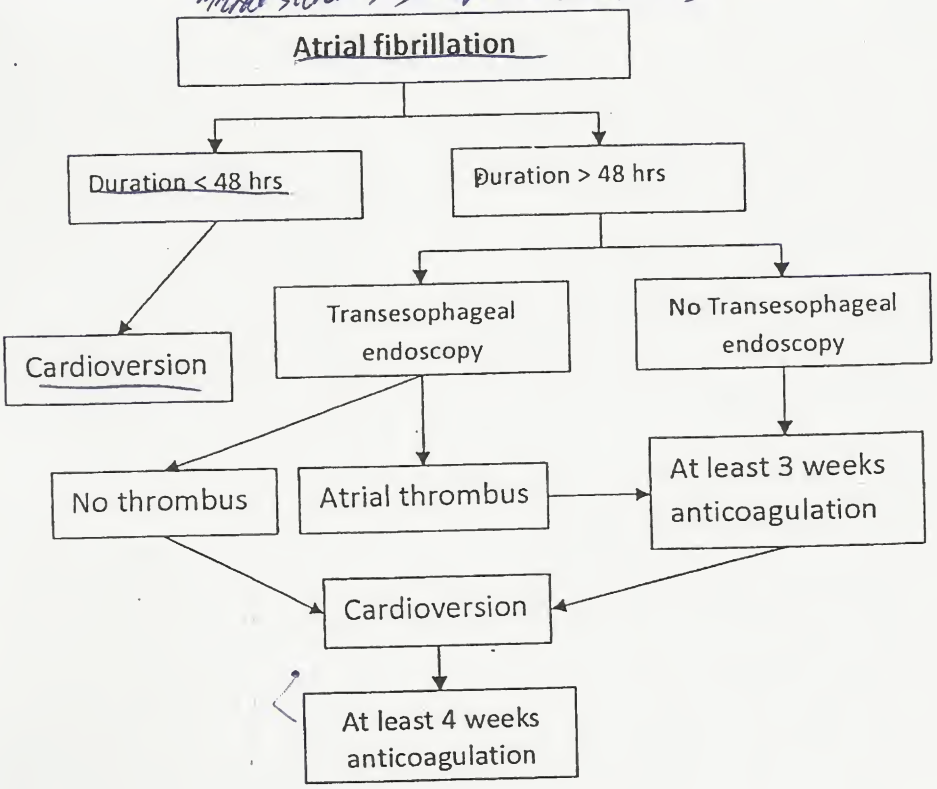
c. If AF is of > 2 days then **Anticoagulation** with Warfarin to an INR of 2-3 for **3 consecutive weeks** should be done before DC cardioversion, because of the risk of embolisation of atrial thrombus and following cardioversion anticoagulants must be continued for at least **4 weeks** until mechanical function returns to normal.

d. In patient with failed DC cardioversion then another electrical cardioversion or chemical cardioversion with Amiodarone or Sotalol can be used.

e. Drugs for prevention of recurrence of AF are Amiodarone (class III) is drug of choice for patients with structural heart disease, & flecainide (class Ic) is drug of choice in patient without cardiac disease. [Ablation therapy may be used]

3- **Prevention of Thromboembolism** in pt with permanent AF:

- Both Aspirin & Warfarin reduce the risk of stroke [pt with AF have 5 times higher risk or stroke]
- If pt is < 65 years & there is no structural heart disease give aspirin
- If pt is > 65 years or there is structural heart disease give warfarin



# Junctional tachycardia or Paroxysmal supraventricular tachycardia

- The problem is → each atrial beat → ventricular beat because AVN is bypassed [Vent rate 150-250]

## 1- AV Nodal Re-entry Tachycardia (AVNRT)

Definition: It is a reentry circuit forming just next to or within the AV node itself.

*Management*

- If the patient is unstable→ DC shock
- Maneuvers that ↑ the vagal tone [Carotid artery message, Valsalva]
- If patient is stable **IV Adenosine** is the drug of choice to restore rhythm
- If frequent → prophylactic prevention with *β-blockers, CCB, or Digoxin*
- Catheter ablation results in complete cure

## 2- Atrioventricular Re-entrant Tachycardia (AVRT)

### A. WPW [Wolff-Parkinson-White syndrome]

It is a reentry circuit with one portion of the circuit is usually the AV node, and the other, an abnormal accessory pathway from the atria to the ventricle.

When the accessory pathway→ if symptomatic it is called WPW.

*Types:*

- **Orthodromic tachycardia:** The re-entry circuit passes antegradely through the AV node & retrogradely through the accessory pathway → narrow-QRS tachycardia
- **Antidromic tachycardia:** The re-entry circuit passes antegradely through the accessory pathway & retrogradely through the AV node → wide-QRS tachycardia.

*ECG*: Premature activation of ventricular tissue via the pathway produces a short PR interval and a 'slurring' of the QRS complex, called a delta wave, the QRS complex may be narrow or wide.

*Management*

- If the patient is unstable→ DC shock
- Maneuvers to ↑ the vagal tone [Carotid artery message, Valsalva] treat ¼ of cases
- If patient is stable **IV Adenosine** is the drug of choice
- If frequent and symptomatic → Prophylactic prevention with flecainide, propafenone or amiodarone.
- Note: *Calcium channel blocking, or Digoxin* are contraindicated because they block the AV node and allow the transmission through the abnormal pathway
- **Catheter ablation is the treatment of choice**

### B. Lown-Ganong-Levine syndrome

An accessory tracts connect the atria directly to the proximal bundle of Hiss.

**ECG** shows a short PR interval (<0.12s) without (no delta waves).

**Treatment** is the same as for WPW syndrome.

# Ventricular Arrhythmias

Ventricular rhythm is characterized by **wide QRS** complex (>0.12 msec)

***Ventricular Ectopic beats (VEBs) = Ventricular Extra-systole***

Ventricular ectopic beat (VEB) are beats which not preceded by a P wave and have a broad QRS.

Frequent VEBs are not significant in pt without heart disease but Frequent VEBs are **poor prognostic** features in pt with **Acute MI or heart failure.**

## Definitions

- *Bigeminy*: in which every sinus beat is followed by a VEB.
- *Trigeminy*: in which two sinus beats are followed by a VEB.
- *Couplets: Pair of* VEB.
- *VT (ventricular tachycardia):* Three or more consecutive VEBs with rate >100 beats/min
- *VF (ventricular fibrillations):* bizarre shaped QRS

## *Ventricular tachycardia*

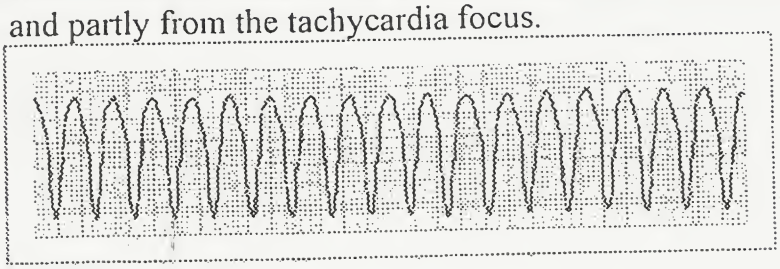
*Causes*: as above but mainly cardiac causes.

Physical examination may show canon a waves

*ECG*: Three or more consecutive wide QRS complexes with rate >100 beats/min, there is no P waves and T waves are inverted. Presence of capture beat and fusion beats are diagnostic of VT. regular R R interval

**Capture beat:** it is when a P-wave that occurs in time at which it becomes conducted → normal QRS morphology without interrupting the tachycardia.

**Fusion beat:** occurs when activation of the ventricle is partly via the normal His–Purkinje system and partly from the tachycardia focus.



**Management:**

- If the pt is unstable → DC shock
- If the patient is stable then drug of choice is Amiodarone, followed by procainamide, & lidocaine.
- VT in patients with structural heart [Post MI, or HF] disease is treated with the implantation of an ICD to prevent VT recurrence

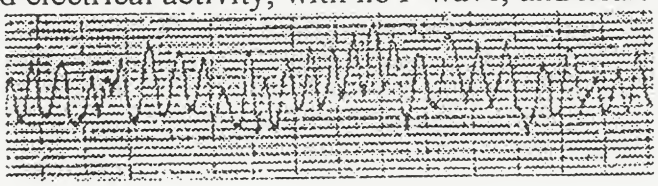
Implantable cardiac defibrillator

verapamil is absolute contraindicated

Torsades de Pointes:
It a special form of VT which occurs in the presence of Long QT interval. The ventricular rate is (250-350 b/min) Its treated by **intravenous magnesium.**

## *Ventricular Fibrillation*

Ventricular fibrillation is characterized by a lack of ordered contraction of the ventricles; therefore, there is no cardiac output. Thus, ventricular fibrillation is synonymous with death unless conversion to an effective rhythm can be accomplished. The ventricular rate is (350-450 b/min)
*ECG*: Bizarre shaped electrical activity, with no P wave, and heart rate 300-600



**Cardiac arrest** = collapsed and pulseless pt = start ABC
*Causes*:

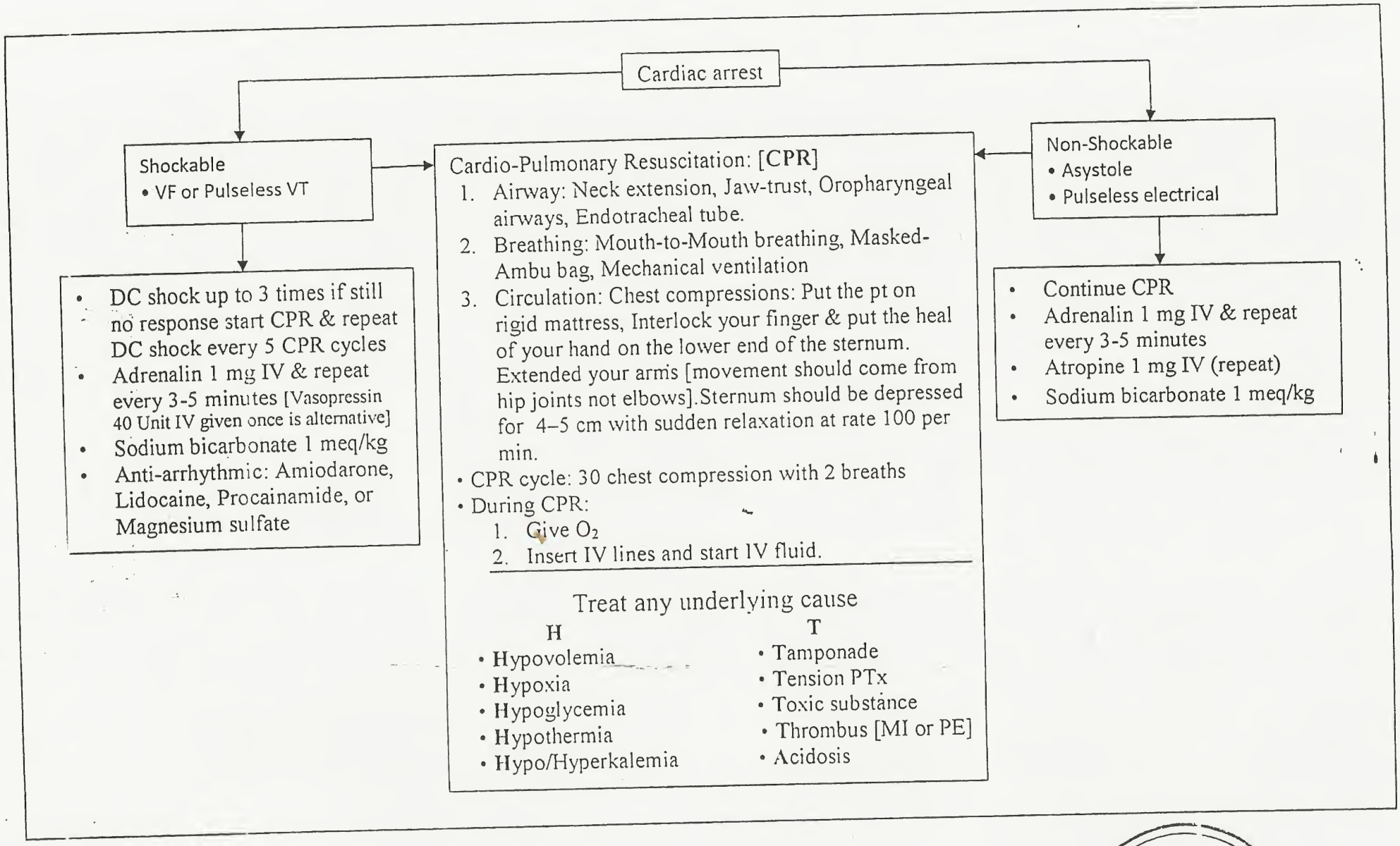
1. Asystole: no QRS complexes [most common cause in children]
2. Pulseless electrical activity (PEA): QRS complexes but no palpable pulse
3. Ventricular fibrillation or tachycardia [most common cause in adults]

Management:

- **Basic life support = CPR:** is aimed to maintain organ perfusion until definitive treatment is done. The CPR includes the lungs ventilation & chest compression. Mouth-to-mouth respiration is used if no equipment is available (oropharyngeal airways, masked Ambu bag).

• In CPR the lungs are inflated 2 times in succession every 30 chest compressions.

- **ALS ( Advanced life support):** is aimed to achieve adequate ventilation, control arrhythmias, stabilize BP & cardiac output. The activities carried out to achieve these goals include:
  1. Defibrillation/cardioversion and/or pacing.
  2. Intubation with an endotracheal tube.
  3. Insertion of an intravenous line.
  - Immediate defibrillation should precede intubation & insertion of an intravenous line; CPR should be started while the defibrillator is charging. As soon as a diagnosis of VF or VT is established, a shock of at least 300 J should be delivered. Additional shocks, up to a maximum of 360 J, are tried if the initial shock are not successfully.



# Antiarrhythmics

There are five main classes in the Vaughan Williams classification of antiarrhythmic agents:

- Class I agents interfere with the sodium ($Na^+$) channel.
- Class II agents are anti-sympathetic nervous system agents. Most agents in this class are beta blockers.
- Class III agents affect potassium ($K^+$) efflux.
- Class IV agents affect calcium channels and the AV node.

Note: Antiarrhythmic drugs not included in this classification digoxin and adenosine

| Class | Examples | Mechanism | Clinical uses | Side effects |
|---|---|---|---|---|
| Ia | Procainamide | $Na^+$ channel blocker | Convert/prevent AF/VT | Nausea, lupus-like syndrome, agranulocytosis, QT prolongation |
| Ib | Lidocaine<br>phenytoin | | VT | Confusion, seizures, respiratory arrest |
| Ic | Flecainide<br>Propafenone | | Prevention paroxysmal AF | Exacerbation of ventricular arrhythmia, prolongation of PR and QRS intervals<br>Propafenone lead to bronchospasm |
| II | Propranolol<br>Metoprolol | β-blockers | Treatment and prevention of SVT and AF/AFL | CHF, bradycardia, AV block, bronchospasm |
| III | Amiodarone | $K^+$ channel blocker | AF, AFL, SVT, VT/VF | Thyroid abnormalities, pulmonary fibrosis, hepatitis, corneal microdeposits, bluish skin, confusion, photosensitivity, QT prolongation |
| IV | Verapamil<br>Diltiazem | $Ca^{2+}$ channel blocker | Prevention of SVT due to AV nodal reentry<br>AF/AFL rate control | AV block, CHF, hypotension, constipation |
| Unclassified | Digoxin | Na-K ATPase blocker | AF/AFL rate control | AV block, ventricular or supraventricular arrhythmia, confusion, anorexia, yellow vision |
| | Adenosine | adenosine → ↓Ca | Terminate reentrant SVT involving AV node | Facial flushing |

| Narrow-QRS complex Tachyarrhythmias | | | |
|---|---|---|---|
| **Arrhythmia** | **Atrial rate** | **Ventricular rate** | **Treatment** |
| Sinus Tachycardia | 100-150 | 100-150 | Rx cause ± β-blocker |
| Atrial flutter | 250-350 | 100-150 | • If the pt is unstable DC shock<br>• Maneuvers to ↑ vagal tone [carotid message or Valsalva]<br>• Rate control by β-blocker, CCB, or Digoxin<br>• Revision to sinus rhythm by DC shock or Drugs [Amiodarone or Sotalol]<br>• Prophylaxis with Amiodarone or Flecainide |
| Atrial Fibrillations | 350-600 | 100-150 | |
| AVNRT & AVRT | 150-250 | 150-250 | • If the pt is unstable DC shock<br>• Maneuvers to ↑ vagal tone [carotid message or Valsalva]<br>• If the pt is stable Adenosine |

| Wide-QRS complex Tachyarrhythmias | | |
|---|---|---|
| **Arrhythmia** | **Ventricular rate** | **Treatment** |
| Ventricular Tachycardia | 150-250 | • If the pt is unstable DC shock<br>• If the pt is stable: Amiodarone, Procainamide, or Lidocaine |
| Torsade de point | 250-350 | • If the pt is unstable DC shock<br>• Magnesium sulfate |
| Ventricular Fibrillation | 350-450 | • DC shock<br>• CPR with 30 chest compression 2 breaths<br>• Adrenalin every 3-5 minutes<br>• Anti-arrhythmic: Amiodarone, Lidocaine, Procainamide, or Magnesium sulfate |

# CORONARY HEART DISEASE [CHD] = ISCHEMIC HEART DISEAS [IHD]

*Ischemia:* lack of oxygen due to inadequate perfusion of a tissue→ imbalance between oxygen supply and demand.
*Ischemic heart disease* (IHD): is a condition in which there is an imbalance between oxygen supply and demand to a portion of the myocardium.

*Atherosclerosis:* Atherosclerosis is a progressive inflammatory disorder of the arterial wall that is characterised by **focal** lipid-rich deposits of atheroma that remain clinically silent until:

1. Atheroma becomes large enough to impair arterial perfusion (i.e. ischemia).
2. Ulceration (discontinuation of the intimal layer over atheroma) leading →
   a. Thrombus formation→ partial or complete obstruction
   b. Embolisation of the overlying thrombus or atheroma material
3. Weakening of the vessel may lead to aneurysm formation

*Atheroma = atheromatous plaque:* a well-demarcated yellow area or swelling on the intimal surface of an artery; and it results from subintimal collections of fat, smooth muscle cells, fibroblast and intercellular matrix.

Atherosclerosis tend to occur at branching points of arteries as at branching point of the Left coronary artery, Carotid bifurcation, Circle of Willis, Proximal renal artery, superior mesenteric artery, and arteries of the lower limbs.

Major risk factors for atherosclerosis are:

1. ↑Age: **Age is the most powerful independent risk factor for atherosclerosis**
2. Male gender and post-menopausal women [because estrogen is protective]
3. Family Hx of **premature** CHD [First-degree relative ♂ < 55yo or ♀ < 65yo]
4. ↑LDL and ↓HDL
5. Cigarette smoking
6. Hypertension
7. Diabetes mellitus
8. Obesity and physical inactivity

- Alcohol if taken in moderate amount 2-4 units [One unit of alcohol is 10ml by volume or 8g by weight, of pure alcohol] will ↓ the risk but excessive intake will increase the risk.

Other risk factors: Type A personality, raised blood levels of **CRP**, & Factors that ↑ the risk of thromboembolism [e.g. ↑level of fibrinogen, ↑ level of homocysteine]

**Coronary heart disease is the most common heart disease and the single most important cause of death.**

Pts with ischemic heart disease fall into two large groups:

1. Pts with chronic coronary artery disease (CAD) = stable angina
2. Pts with **Acute Coronary Syndromes** (ACSs) which is composed of:
   a. Acute myocardial infarction (MI) with ST-segment elevation on ECG
   b. Unstable angina & non-ST-segment elevation MI (UA/NSTEMI).

## Angina pectoris

Its **clinical syndrome** is due to **transient** myocardial ischemia (not necrosis) & this clinical syndrome consists of:

1. An uncomfortable sensation is described as "Pressure, squeezing, compression, tightness, heaviness, burning" it's substernal in location, & may radiate to neck, jaw, shoulders, arms, or epigastrium.
2. The angina is triggered by (1) exertion, (2) after meals (3) emotional stress (4) exposure to cold
3. Angina is relieved by rest or nitroglycerin (within < 10 minutes).
4. It **may** be associated with dyspnea, diaphoresis, nausea, or vomiting.

**Stable angina**: Its angina that occurs in a well-defined, reproducible pattern-usually on exertion.

**Unstable angina** refers to angina with one of the following features:

(1) crescendo angina, defined as previously diagnosed angina that has become more frequent, longer in duration, or more severe in nature

(2) new onset angina (within 2 months) brought on by minimal exertion

(3) rest angina of >20 minutes in duration

(4) post-MI angina (occurring >24 hours after MI)

= CAD
= ECG

| Condition | Duration | C/P | Associated Features |
|---|---|---|---|
| Stable Angina | > 2 & <10 min | Angina pectoris | ↑ by exertion, cold, stress, relived by nitroglycerine or rest. |
| Unstable angina | 10–20 min | Similar to angina but often more severe | Similar to angina, but occurs with low levels of exertion |
| Acute MI | > 30 min | Similar to angina but often more severe | **Unrelieved by nitroglycerin or rest** |

Spectrum of Coronary artery diseases:

- Stable angina • Unstable angina/NSTEMI • STEMI
- Heart failure either due to Ischemic cardiomyopathy or Post MI
- Arrhythmias due to ischemia

| | Stable angina | Unstable angina | NSTEMI | STEMI |
|---|---|---|---|---|
| Mechanism | Stenosis of > 75% of the supplying vessel | Stenosis of > 90% of the supplying vessel | Near total Occlusion of the supplying vessel→ Subendocardial infarction | Total Occlusion for >30min of the supplying vessel→ Transmural infarction |
| C/P | See above | See above | See above | See above |
| ECG | • Resting is normal<br>• Exercise is abnormal | • ST segment depression &/or<br>• New T wave inversion<br>• No Q-wave | | • ST segment elevation<br>• Q-wave |
| Cardiac enzymes | No elevation | No elevation of cardiac enzymes after 12 hours | Elevation of cardiac enzymes | Elevation of cardiac enzymes |
| Treatment | Rx Risk factors<br>Aspirin<br>Nitrates<br>β-blockers or CCB | Rx Risk factors<br>Aspirin + Clopidogrel<br>Unfractionated or LMW heparin<br>Nitrates<br>β-blockers or CCB<br>**Do not administer Thrombolytic therapy** | | Rx Risk factors<br>Aspirin + Clopidogrel<br>Unfractionated or LMWH<br>Nitrates<br>β-blockers or CCB<br>Thrombolytic therapy |

**NSTEMI**: symptom of unstable angina + ECG [ST segment depression ± T-wave inversion] + Elevated cardiac enzyme

NSTEMI = Subendocardial MI = Non-Q wave MI

**STEMI**: MI pain + ECG [ST elevation] + Elevated cardiac enzyme

STEMI = Transmural MI = Q-wave MI

The angina occurs when there is a mismatch between oxygen demand & the oxygen supply

D.D of angina:-

- anemia
- pulmonary embolism
- arrithmia
- aortic valve disease

Angina

Sudden sever central squizing or crushing chest pain, It may radiate to inner aspect of the arm, Jaw & the root of the neck

Low Risk

High Risk

- L.A coronary block or 3 lesser vessels
- angina with small work load
- strongly + stress ECG
- post infarct angina
- left vertricular hypertrophy

# Stable angina

***Clinical picture***: (see above for anginal pain description)

Special forms of angina:
**Decubitus angina:** Is that occurring on lying down. It usually occurs with association with impaired left ventricular function.
**Nocturnal angina:** Is that occurring at night and may wake up the pt from sleep. It may be provoked by vivid dreams
**Angina equivalent**: symptoms of LVF (dyspnea) indicating presence of ischemia

***Examination***

- Often normal
- Look for signs of anemia & hyperthyroidism
- During episode of angina:
  - Transient S3 or S4
  - Transient murmur of mitral regurgitation (papillary muscle ischemia)
  - Transient basilar rales

***Investigations***

**Resting ECG**:

- In absence of pain its normal in 50% of pts. May show evidence of previous MI
- During the anginal attack may show ischemic changes [ST-segment depression]

**Stress testing**:

1. Exercise ECG:
   - By using a standard treadmill or bicycle while monitoring the patient's ECG, BP.
   - Resuscitation facilities should be immediate to hand.
   - *Indications for exercise ECG:*
     1. To **confirm the diagnosis of angina**
     2. To evaluate stable angina
     3. To assess prognosis following myocardial infarction
     4. To assess outcome after coronary revascularization (PCI or CABG)
   - The test should be stopped immediately if the patient develops:
     (1) Chest discomfort (2) Severe Dyspnea (3) Dizziness (4) Lowering of Systolic BP > 10 mmHg (5) ventricular arrhythmias (6) ST segment depression > 2 mm
   - **Positive test criteria**: **Flat or down-sloping ST segment depression** ≥ 1 mm lasting for more than 0.08 msec is indicative of ischemia
   - **High risk criteria** (are associated with Poor prognosis & are indication for coronary angiography):
     1. Low threshold for ischemia (i.e. stage 1 or 2 of the Bruce Protocol)
     2. Fall in BP on exercise
     3. Ischemic changes occurring in > 5 ECG leads
     4. Exercise-induced arrhythmia

- **Contraindications** to Exercise ECG:
    1. In pts with acute MI within 2 days
    2. Unstable angina until medically stabilized
    3. Severe aortic stenosis
    4. Uncontrolled hypertension
    5. Uncontrolled HF
    6. Active pericarditis, myocarditis, endocarditis
- Stress ECG sensitivity is 75% → -ve stress ECG does not exclude CAD

2. Stress Echo or Stress scintigraphy are done in pts who are having abnormal baseline ECG e.g. WPW, LBBB.

**Echocardiography:** Can assess valve causes of chest pain (AS & MVP), and can assess LV function

**Coronary angiography** (see below)

## *Management*

- Prevent the progression of CAD
- Optimize life expectancy
- Relieve symptoms

**Prevention of the progression of CAD:**

This is done by reducing the risk factors

1. Weight reduction, and exercise 30 minutes >4x/week
2. Stop smoking
3. Aspirin prevents thromboembolism in coronary circulation resulting in lower risk of MI → **improving the prognosis** [Aspirin has no effect on symptoms]
4. ACEI which improve prognosis in HF & Post MI
5. Statins for hyperlipidemia to keep **LDL <100 and HDL > 35**
6. In hypertensive Control **BP to < 135/ 85 mmHg**
7. In DM pt: **FBS <120, HA1c <8**

**Optimizing life expectancy** (see below)

Life expectancy can be improved by coronary revascularization in selected cases

**Relieve of Symptoms**

The four main drugs have additive effect and used in stepwise. [Some Pts with mild angina are reluctant to take maintenance therapy and prefer to use sublingual nitrate as needed] and all of them are similar in effectiveness in relieving the symptoms

- First step → β-blocker
- 2nd step → Calcium antagonist
- 3rd step → Nitrate
- If combination of 2 drugs fails to control symptoms revascularization should be considered

## ➢ β-blockers

*Mechanism of action:*

↓ $O_2$ demand by reducing heart rate, blood pressure and myocardial contractility

Note: A β-blocking drug should not be withdrawn abruptly because this may have a rebound effect and precipitate dangerous arrhythmias, worsening angina or MI.

## ➢ Calcium antagonists

*Mechanism of action:*

- Arteriolar dilatation → ↓ afterload → ↓ contractility → ↓ O2 demand
- Directly on myocardium → ↓ contractility
- Directly on the AV node → ↓ rate

Dihydropyridine calcium antagonists, (such as nifedipine and nicardipine) because they can act on peripheral blood vessel in addition to their action on the heart they often cause a reflex tachycardia; so use these drugs in combination with a β-blocker.

**In contrast** verapamil and diltiazem are particularly suitable for patients who are not receiving a β-blocker because they act selectively on the heart.

*Side effects:*

- May precipitate heart failure because they ↓ contractility.
- Peripheral edema.
- Flushing, headache & dizziness.

## ➢ Nitrates

*Mechanism of action:*

- Venodilatation→ ↓ preload & arteriolar dilatation→ ↓ Afterload [both ↓ $O_2$ demand of the heart]
- Coronary vasodilatation→ ↑ $O_2$ supply to the heart.

*Side effects:*

- Headache
- Hypotension –rarely→ syncope
- Hypotension may lead to reflex tachycardia

**Note:** To avoid these symptoms the tablet may be spat out as soon as the angina is relieved.

- Pts are advice to use the drug prophylactically before engaging in exercise that is likely to provoke symptoms.
- **Nitrate tolerance** develops if they are given throughout the 24 h, so a "**nitrate-free**" period of 6-8 hours is needed

Routes of nitrates:

- Sublingual nitrates, it is given sublingually because it has high first-pass metabolism [If taken orally it gets metabolized in liver & does not reach the systemic circulation]
- GTN Transcutaneous patch

## Coronary angiography Indications:

1. High risk stress ECG changes
2. Symptoms uncontrolled by the use of 2 anti-anginal drugs
3. Convincing history with –ve stress ECG results
4. Post infarct angina
5. HF with angina — angina with going variant

## Coronary Revascularization therapy

1- Percutaneous coronary intervention (PCI): by the use of balloon & stent

*Indication*: the patient is having single or 2 vessels disease

- PCI provides an effective symptomatic treatment but there is **no evidence that it improves survival** in patients with chronic stable angina.

2- Coronary artery bypass graft (CABG) surgery is *indicated* when it's shown to **improve the prognosis** which is in:

1. ALL 3 coronary arteries (RCA, LAD, LCX)
2. The left main coronary artery
3. Pts with DM

- Arterial graft (usually using internal mammary artery) is superior to venous grafts (usually grater saphenous vein)

## Angina with normal coronaries

***Prinzmetal's angina* = *Variant angina (Coronary vasospasm)***

*C/P:* Chest discomfort is similar to angina but more severe and occurs typically at rest.

*Pathophysiology:* Intermittent focal spasm of coronary artery; it's **often** associated with atherosclerotic lesion near site of spasm. But it may occur in normal coronaries.

*Investigations:* ECG (ambulatory Holter monitor) for **transient ST elevation**; diagnosis confirmed at coronary angiography using provocative IV acetylcholine test.

*Treatment:* Longacting nitrates and calcium antagonists. [Not β-blockers] responds well to ~~not~~ antianginal treatment

*Syndrome X*

- Typical angina on effort + ischemic changes on stress ECG testing + angiographically normal coronary arteries.
- The etiology is not known but prognosis is good.

# Unstable angina & NSEMI Management

**Clinical picture: see above**
**Physical examination: as stable angina**

**ECG** Most commonly ST depression and/or T-wave inversion; no Q-wave.
**Cardiac biomarker:** CK-MB and/or troponins are elevated in NSTEMI. But not UA

## *Management*

### ➢ Immediate Management

**Antithrombotic Therapy:** Aspirin + Clopidogrel + Heparin
- IV GP IIb/IIIa antagonist [Tirofiban, Eptifibatide] for high-risk pts for whom invasive management is planned
- All of these Antithrombotic treatments reduce the mortality
- **Do not administer thrombolytic therapy to pts with UA/NSTEMI.**

**Invasive vs. Conservative Strategy**
In high-risk pts [defined as **Any** of the following: Recurrent ischemia, ↑ serum troponin, ST segment ↓, LVF, ↓BP, VT or prior CABG, an early coronary arteriography within <48 hr followed by PCI or CABG improves outcomes.

### ➢ Long-Term management

1. **Beta blockers**.
2. **Statins**
3. **ACE inhibitors** are recommended for long-term plaque stabilization.
4. Antiplatelet therapy combination of **Aspirin** and **Clopidogrel** for at least 9–12 months, with aspirin continued thereafter.

## *Prognosis*

- 15% of patients with UA/STEMI progress to STEMI or Death
- 1/3 will suffer a recurrence of severe ischemic pain, within 6 months of index event

# ST-Elevation Myocardial Infarction [STEMI]

***Definition:*** It is an acute coronary syndrome in which there is persistent, complete occlusion of the involved coronary artery. & the pain may last for hours

## *Clinical picture*

- Most pts present with severe, persistent (> 30 min), substernal chest pain associated with nausea, **vomiting, diaphoresis**, dyspnea, and apprehension.
- **Angor animi**: the sense of being in the act of dying. leads to sympathetic stimulation
- About **15%** of pts are Asymptomatic = Painless or silent MI and its most common in older or diabetic pts.
- Symptoms of congestive heart failure (CHF) due to large infarctions.
- Collapse = Syncope which may be due to:
  - Arrhythmias
  - Sever hypotension (cardiogenic shock)
- Sudden death, from ventricular fibrillation or asystole.

> VF is the most common cause of death in the first hour in pt with MI

## *Physical examination*

Many pts have a normal pulse rate & BP within the first hour of STEMI but:

- ½ of Patients with an **inferior MI** will have parasympathetic hyperactivity (bradycardia and/or hypotension ± vomiting).
- ¼ of Patients with **anterior MI** will have sympathetic hyperactivity (tachycardia and/or hypertension).

Fever may be found and it due to tissue damage

Pts may show signs of left or right ventricular failure (see HF sheet)

Precordial examination:

- Diffuse apical impulse
- S3 or S4
- Mitral regurgitation (papillary muscle ischemia)

## *Investigation*

- Blood tests
- ECG
- Cardiac enzymes
- CXR
- Echocardiography due to stress

**Dx of MI** at least 2 of the following 3:

1. Chest pain persisting > 30 min
2. Typical ECG finding
3. Elevated cardiac enzyme

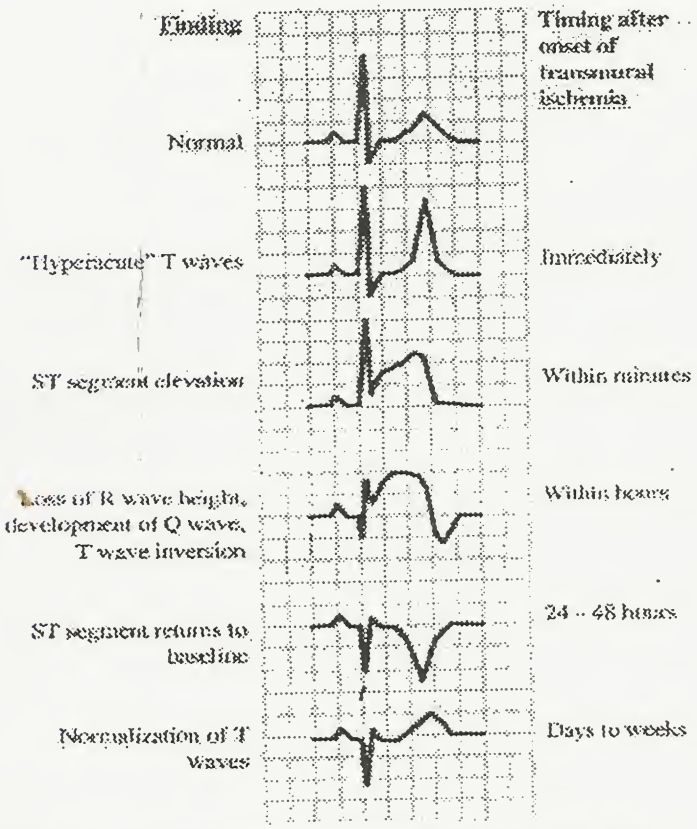
➢ Blood tests: Leucocytosis, ↑ ESR, & ↑ CRP all may occur in MI

## ➢ ECG

The ECG is diagnostic 85% of cases. The remaining 15% of MI don't have clear-cut evidence on the ECG.

**Definition of ECG changes** earliest sign

- *Peaked T wave* (amplitude >6 mV in limb leads **OR** > 10 mV in precordial leads)
- *ST elevation* (amplitude > 1mm in limb leads or >2mm in precordial leads)
- *Pathologic Q waves* (>0.04 s **AND** >25% of total QRS height)




Correlation between Coronary arteries anatomy, & ECG changes:

| Heart wall | ECG changes | Coronary artery |
|---|---|---|
| **Anteroseptal** | V2-V4 | Left anterior descending |
| **Lateral** | I, aVL +/- V5-6 | Left circumflex |
| **Anterolateral** | V2-6, I, aVL | Left main stem |
| **Inferior** | II, III, aVF | Right coronary, marginal branch |

In Inferior MI right sided chest leads should be put to look for right vent.infaretion.

Posterior MI will show tall R wave in leads V1, & V2. It's due to closure of posterior descending artery.

## ➢ Cardiac enzymes

- Myoglobin is the first to rise
- CK starts to rise at 4-6 hours, peaks at about 12 hrs and falls to normal within 48-72 hrs. CK is also found in skeletal muscle, but CK-MB is mainly cardiac.
- Small rise in CK (**not CK-MB**) occurs in: (1) IM injection (2) vigorous physical exercise (3) post trauma (4) Defibrillation.
- CK-MB is useful to look for reinfarction as it returns to normal after 2-3 days (troponin T remains elevated for up to 10 days)
- The **most sensitive** markers of myocardial cell damage are the cardiac **troponins T and I**, which are released within 4-6 hours and remain, elevated for up to 10 days.
- Small troponin elevations may occur in: (1) CHF (2) Myocarditis (3) PE.

Sensitivity Troponins T & I > sensitivity of CK-MB > sensitivity of CK

| | Begins to rise | Peak value | Returns to normal |
|---|---|---|---|
| **Myoglobin** | 1-2 hours | 6-8 hours | 1-2 days |
| **CK** | 4-6 hours | 12 hours | 2-3 days |
| **Trop I** | 4-6 hours | 12-24 hours | 7-10 days |
| **Trop T** | 4-6 hours | 12-24 hours | 10-14 days |
| **AST** | 12-24 hours | 36-48 hours | 3-4 days |
| **LDH** | 24-48 hours | 72 hours | 8-10 days |

2 sets of normal troponins 4-6 hours apart exclude the diagnosis of MI

## ➢ Chest X-Ray

- CXR may give clues to an alternative Dx (aortic dissection or pneumothorax)
- CXR may show complications of MI such as heart failure

## ➢ Echocardiography

- Abnormalities of wall motion are usually present. But acute STEMI cannot be distinguished from an old myocardial scar or from acute severe ischemia.
- It is especially useful when the ECG is not diagnostic of STEMI, and it can aid in management decisions, such as whether the pt should receive reperfusion therapy [e.g., fibrinolysis or a percutaneous coronary intervention (PCI)].

morphine, Oxygene, Nitrate, Aspirine
MONA

## Management

1. CALL FOR HELP
2. Strict rest
3. *On suspicion of acute MI* give **Aspirin** immediately (300 mg chewed) therapitic dose
4. **O2** to correct any hypoxia due to LVF
5. Insert 2 IV cannula
6. Relieve pain with IV **morphine** or diamorphine & antiemetics (metoclopramide) [Avoid IM injections because poor skeletal muscle perfusion delays onset of action and if thrombolytic therapy will be given it will → painful hepatoma].
7. Nitroglycerin sublingually [ If chest discomfort persists after three doses given 5 min apart, consider IV until symptoms relieved or systolic bp < 100 mmHg]
   - Do not use nitrates in pts with recent use of phosphodiesterase inhibitors for erectile dysfunction (sildenafil or tadalafil).
8. Subcutaneous Heparin ↓ risk of reinfarction after successful thrombolysis and reduce the risk of thromboembolic complications.
9. *β- blockers:* ↓ myocardial $O_2$ consumption (relieve pain), limit infarct size, and reduces mortality. "metoprolol"
10. *ACE inhibitors* reduce mortality in pts following acute MI and should be prescribed within 24 h of hospitalization for pts with STEMI. ACEI should be continued indefinitely in pts with CHF or asymptomatic pt with LVEF <40% LV risk of aneurism form
11. Reperfusion (**Time = Muscle**)
    - **Thrombolytics therapy** = Fibrinolysis:
      - They can reduce the hospital mortality of myocardial infarction by 25%-50% and this survival advantage is maintained for at least 10 years.
      - **Streptokinase** or **Alteplase** (human tissue plasminogen activator or tPA) (both given IV infusion), Streptokinase is a foreign protein so may → allergic reaction & hypotension, but Alteplase is recumbent protein so → not allergic.
      - *Side effects*: Bleeding & Reperfusion arrhythmias
      - Note: tPA have better survival rates than streptokinase but with higher risk of intracerebral bleeding.
    - **Primary percutaneous coronary intervention (PCI)**
      - Percutaneous coronary intervention **is the treatment of choice if readily available**. In comparison to thrombolytic therapy, it is associated with a 50% greater reduction in the risk of death, recurrent MI or stroke.
      - PCI is also indicated in pts in which thrombolytic therapy is contraindicated or fails to achieve coronary arterial reperfusion.

Pain Control → Nitrate, Morphine, β-blockers

Limit Infarction size → β-blockers, Reperfusion → Thrombolytics, PCI

MCQ

| Criteria for Thrombolysis in Acute MI |
|---|
| **Indications:**<br>1. Chest pain consistent with AMI, **and**<br>2. ST-elevation > 0.1m in limb leads or > 0.2mm in precordial leads, in at least 2 contiguous leads or new LBBB, **and**<br>3. < 6 hours from onset of symptoms (up to 12 hours if pain persists)<br>4. Patient is younger than 75 years (greater risk of hemorrhage) |
| Absolute Contraindications:<br>1. Active internal bleeding (excluding menses)<br>2. Suspected aortic dissection<br>3. Prior hemorrhagic stroke at any time; & ischemic stroke in the last year<br>4. Known intracranial neoplasm |
| Relative contraindications:<br>1. Uncontrolled HTN on presentation (BP > 180/110 mmHg)<br>2. History of prior CVA<br>3. INR > 2<br>4. Recent trauma (within 2-4 weeks)<br>5. Recent internal bleeding<br>6. Active proliferative diabetic retinopathy<br>7. Pregnancy<br>8. Active peptic ulcer<br>9. H/O chronic sever hypertension<br>10. For streptokinase: prior exposure or prior allergic reaction |

## *Secondary prevention*

- For pts who have not already undergone coronary angiography and PCI, submaximal exercise testing should be performed prior to or soon after discharge, to see if the pt need coronary angiography.
- **Modification of cardiac risk factors**: stop smoking; control hypertension, diabetes, and serum lipids, and exercise.
- **Drugs**:
  - **β-blockers**: should be prescribed for at least 2 years following acute MI.
  - **Statins**
  - **Aspirin** with clopidogrel.
  - If the LVEF <40% an **ACE inhibitor** should be used indefinitely.

## *Return to normal life*

- Mobilization in 2 days in absence of HF
- The usual duration of hospitalization for an uncomplicated STEMI is about 5 days.
- During the first 1–2 wks, pt should be encouraged to ↑ activity by walking about the house and outdoors in good weather. Normal sexual activity may be resumed.
- Most patients will be able to return to work within 2–4 weeks.
- Car driving after 4 weeks

| Treatment | Effect |
|---|---|
| Aspirin | • Aspirin improves short term mortality (30% reduction)<br>• combination of aspirin with clopidogrel adds another 10% reduction in short term mortality<br>• Long term mortality → 25% ↓ in risk of recurrent MI, stroke, or cardiovascular mortality |
| Thrombolytics | • Reduction of both short- and long-term mortality rates & Reduces infarct size by 25-50% |
| β-Blockers | • Improve short-term survival<br>• Improves long-term survival |
| ACEI & ARB | • Improves long-term survival |
| Heparin | • Small reduction in short-term mortality |
| Nitrates & CCB | No effect on mortality |

## *Complications* [Electrical or Mechanical]

Immediate/hours

- Ventricular arrhythmias (VT or VF) are the main cause of death in the first 24 hours therefore Pts should be monitored close to a defibrillator.
- Atrial arrhythmia (atrial flutter, or atrial fibrillation)
- Heart block
- Cardiogenic shock
- LVF: Treatment is with Diuretics, Nitrates, ACEI
- RVF: Treatment is with infusion of colloid to increase preload. [Diuretics & Nitrates worsen the symptoms and therefore are **not** indicated]

Hours/days: Cardiac rupture usually occurs **2-5 days** following MI & may by:

1. Ventricular Septal Rupture (VSD)
2. Papillary muscle rupture → sever MR
3. Cardiac wall rupture into pericardium

Days/weeks

- Thromboembolism
- Ventricular remodeling which worsens the condition → Chronic HF
- **Ventricular aneurysm → sustained ST-segment elevation**
- Ventricular tachycardia may occur & when frequent ICD is indicated
- **Dressler's syndrome**: it's a self limiting autoimmune pericarditis occurs several weeks after a full-thickness MI, treated by NSAID

***Bad prognostic signs in STEMI***: • Hypotension or Marked LVF • Extensive ECG changes • Acute hyperglycemia or H/O DM • ↑ Urea • Old age • LBBB • Anterior infarcts • High cardiac enzyme levels • **Depression** & social isolation

***Surgery and the risk of MI***: Patients with a history of UA or MI elective non-cardiac surgery should be avoided for 3 (and preferably 6) months after such an event. Antiplatelets and **β-blockers** reduce the risk of perioperative MI in Pts with CAD

Valvular disease due to R.F

Mitral → 50%. Mitral & Aortic → 40%. Mitral, Aortic & Tricuspid → 4%.

Aortic → 2%.

Mitral stenosis will lead to ↑ interatrial pressure in the Lt At which in return it will lead to Lt At hypertrophy. this will ↑ the risk of developing A.F "atrial fibrillation" & it may lead to thrombus embolisation.

If the ↑ in interatrial pressure ~~will lead to~~ was sudden than it will lead to pulmonary oedema ~~the~~ in contrast gradual ↑ in interatrial pressure the lungs will compensate in long term ↑ thickness of the wall of pulmonary arteries "pulmonary hypertension"

→ Rt Vt hypertrophy → Rt A hypertrophy → Raised JVP

Symptoms:- -Fatigue ability -dyspnea -palpitation

-chest pain -Cough -haemoptosis

② thromboembolisation:-

-CVA -LL ischemia

③ Lt At hypertrophy:-

-dysphagia -hoarseness of voice

④ Invective endocarditis

---

D.D of cheek pigmentation:-

-mitral stenosis -hypothyroidism -SLE

-Cushing syndrome -Rosetia -freckles

# Valvular heart diseases

## Mitral Stenosis

The normal mitral valve area is 4.0-6.0 $cm^2$ & pt become symptomatic when < 2 $cm^2$
Mild stenosis→ 1.5-2.5 $cm^2$
Moderate stenosis→1.0-1.5 $cm^2$
Severe stenosis → <1.0 $cm^2$

### *Epidemiology*

- Age: usually present at age 40-50 years.
- Gender: more in **Females**

### *Etiology*

1. **Rheumatic fever** MOST COMMON CAUSE
2. Congenital abnormalities (rare)
3. Senile degeneration (rare)
4. SLE (rare)

### *Clinical picture*

- **Only half** of the pts will give a H/O RF during childhood.
- Any factor that ↑ cardiac output it will → ↑ in Lt atrial pressure → will make the symptoms worse e.g. exercise or **pregnancy**.

### *Symptoms & Pathophysiology*

Progressive fibrosis & calcification of the valve leaflets→ MS → ↑Lt At pressure →

1- ↑ Pulm. venous pressure →
- **Dyspnea** (most common presenting symptoms)
- Rupture of capillaries →**Hemoptysis**
- Pulm.HTN may → **Chest pain**
- Pulm.HTN → RVH → **RVF** symptom

2- Lt atrial enlargement →
- Pressure on esophagus → **Dysphagia**
- Pressure on Lt recurrent laryngeal Nerve leading to Hoarseness (**Ortner's syndrome**)
- Arrhythmias (mainly **AF**)→ **Palpitations**
- Blood stasis → **Thromboembolism** [CVA, limb ischemia]

### *General examination*

Pulse → **Small volume**, & if AF present→ irregularly irregular
Face → **Malar rash** "cheeks"
Neck →If Pulm. HTN → prominent a wave in JVP. & If RVF → ↑JVP
LL→ if RVF → LLE

### *Precordial examination*

I→ if PHT → pulsation in pulmonary area, & If RVH →Lt parasternal heave.
P→ **Tapping apex beat**, Thrill if murmur ≥ 4 , & if RVH Lt parasternal heave.
A→ Loud S1 & normal S2 or loud + low pitched **rumbling Mid-diastolic murmur** at mitral area heard best with bell in left lateral position with pt in expiration with opening snap. with presystolic accentuation due to contraction

**Opening Snap** High-pitched; follows S2, heard at apex in mitral stenosis
- The more severe the MS, the shorter the S2–OS interval
- When S1 & OS are inaudible it indicates that the valve is heavily calcified.

So the presence of opening snap indicates a mobile relatively good valve.

### *Investigations*

- **ECG:** M shaped
  - P-mitral (Left atrial enlargement), or AF, or RVH.
- **CXR**: may show Left atrial enlargement [Mitralization of lateral border of the heart, Cardiomegaly, & splayed tracheal bifurcation] Kerley B lines.
- **Echocardiography: Initial investigation of choice** because it Confirms the diagnosis & Assesses the severity of the stenosis - measures the size of the valve - structural abnormalities - EF & SV, CO - Diameter of each chamber & its walls

### *Management*

- Treat arrhythmias and HF & AF "B-blocker"
- Prophylaxis for infective endocarditis & Prophylaxis for rheumatic fever [if <25yr] antibiotic
- Asymptomatic pt → follow up by echo every 6-12 mo.
- **Diuretics** to control pulmonary congestion
- **Warfarin** to control thromboembolic complication.
- **Surgery**:
  - Indications:
    - Symptomatic pt
    - Asymptomatic pt with mitral orifice < 1.7 $cm^2$ or with pulmonary Htn
  - Type of Surgery:
    - Percutaneous **balloon valvuloplasty** is the procedure of choice; if not possible, then open surgical valvotomy or replacement.
    - If pt has co-existing MR then replacement.

# Mitral regurgitation

## *Etiology*

1- Dilatation of ventricle [CHF or Idiopathic]
2- Rheumatic fever.
3- Infective endocarditis
4- Post-MI with rupture of papillary muscles
5- Mitral valve prolapse (MVP)

## *Clinical picture*

- Symptoms & sign of LVF ± Symptoms & signs of RVF If Pulm HTN occured
- Precordial examination: Pansystolic murmur best heard over the mitral area with radiation to axilla and it decreases by inspiration.

## *Investigations*

- **ECG:** Left atrial enlargement, AF, RVH, or LVH.
- **CXR:** may show Cardiomegaly due to Left atrium or Left ventricle
- **Echocardiography: Initial investigation of choice** because it Confirms the diagnosis **&** Assesses the severity of the regurgitation

## *Management*

- Treat arrhythmias and HF
- Prophylaxis for infective endocarditis & Prophylaxis for rheumatic fever [if <25yr]
- Asymptomatic pt → follow up by echo every 6-12 months.
- Afterload reduction (ACE inhibitors)→ ↓ regurgitation & ↑ forward cardiac output.
- **Surgery**:
  - Indications:
    - Symptomatic pt
    - Asymptomatic pt: LV dysfunction (LVEF < 60% or endsystolic LV diameter by echo > 45 mm) Operation done *before* development of CHF.
  - Type of Surgery: Repair or Replacement.

# MVP (Barlow's syndrome) =Floppy mitral valve

***Etiology:*** **Most commonly idiopathic**, others causes: Familial, Rheumatic fever, Ischemic heart disease, ASD, and Marfan syndrome. Its more in females

***Clinical picture***: Mostly asymptomatic but some pts present with lateral Chest pain

***Examination*:** Mid-systolic click ± **Late** systolic murmur.

***Complications***

1- Thromboembolic symptoms→ TIA in patient > 45 yrs old [There is no association between MVP & TIA in pts < 45 yrs]
2- Arrhythmias
3- Infective endocarditis [If associated with MR]
4- Sudden death [very rare]

***Management:*** Asymptomatic no Rx. , but if associated with MR Rx as MR. Aspirin or anticoagulants for pts with history of TIA or embolisation.

# Aortic Stenosis

The **Most Common** valve disease [at age of 80 years 10% of population has AS]
Anatomy: The normal aortic valve area is 3.5 $cm^2$. ?

- Mild stenosis→ 1.5-2.0 $cm^2$
- Moderate stenosis→ 1.0 1.5 -$cm^2$
- Severe stenosis→ <1 $cm^2$ (or a mean gradient of>50mmHg)

## *Etiology*

1- **Congenital** (rare).
2- **Premature calcification** of a **congenitally bicuspid aortic valve**: pts typically develop symptoms by age 40 yrs.
3- **Rheumatic fever**: Always associated with mitral valve disease & usually develop in 4th to 5th decade.
4- **Calcific aortic stenosis of normal valve**: The **most comm.** Pt > 65 yrs. "senile"

## Symptoms (*Classical Triad*) S.A.D "Syncope, Angina, Dyspnea"

1- **Effort dyspnea** [due to left ventricular failure]
2- **Effort angina** [severe LVH→ mismatch between O2 supply & O2demand] ± CAD
3- **Effort dizziness or syncope** [due to peripheral vasodilation → hypotension in the presence of a fixed cardiac output.]

- Sudden death [very rare].
- Pts may present with symptoms & signs of LVF

*Examination* Precordial examination→ (see table)

*GE:*

- BP: Narrow Pulse Pressure[ PP < 25% of the systolic value]
- Pulse: Small volume[Pulsus parvus], Slow rising pulse [Pulsus tardus]
- A thrill may be palpable over the carotid or over the supra-sternal notch

*Precordial examination:* Systolic ejection murmur best heard over the aortic area and it radiates to carotids. Pt has paradoxical splitting of the second heart sound.

## *Investigation*

- **ECG**:
  - Left ventricular hypertrophy (usually) Ventricular arrhythmia
  - LBBB (due to calcification of the conducting system)
- **Chest X-ray** → May show LVH or Calcifications of the aortic valve "But usually normal"
  - **Echocardiography**: is the **Initial investigation of choice** because it Confirms the diagnosis & Assesses the severity of the stenosis
    - Size of the valve
    - calcification
    - thrombosis
    - diameter of chambers
    - thickness of the wall
    - EF, EDV, ESV, SV, Co

***Management***

- Avoid strenuous activity in severe AS, even in asymptomatic phase.
- Treat arrhythmias and Treat HF in standard fashion but *avoid afterload reduction [No vasodilators].*
- Prophylaxis for infective endocarditis & Prophylaxis for rheumatic fever [if <25yr]
- Asymptomatic pt → follow up by echo every 6-12 months.
- **Surgery**:
  - Indications:
    - Symptomatic pt [death usually occurs in 3-5 yrs of the onset of symptoms]
    - Asymptomatic pt: with valvular gradient > 50 mmHg (Operation done *before* development of CHF).
  - Type of Surgery:
    - **Valve replacement** is indicated [death usually occurs within 3-5 years of the onset of symptoms].
    - Balloon valvuloplasty is limited to pts with congenital cause or critical aortic stenosis who are not fit for valve replacement
- Statin therapy may slow progression of leaflet calcification.

## Aortic Regurgitation

| Causes | Acute | Chronic |
|---|---|---|
| Dilatation of valve ring | Aortic dissection<br>MI<br>RF | Idiopathic<br>**Ankylosing spondylitis**<br>**Hypertension**<br>Syphilitic aortitis<br>Marfan's syndrome<br>SLE |
| Diseased valve cusps | **Infective endocarditis** | Congenital → bicuspid valve<br>Acquired→ RF, SLE |

**Epidemiology**: More common in males

*Clinical picture*

- Those of LVF [dyspnea, orthopnea, PND, fatigue]
- ☆Angina may occur due to low diastolic pressure reducing coronary perfusion

*GE:*

- *BP:* Wide Pulse pressure [PP > 50% of the systolic value] "collapsing pulse"
- *Pulse:* Large volume & Bounding = Collapsing pulse [*Pulsus bisferiens*: Double systolic pulsation felt in carotid occurs in mixed AR & AS].

***Precordial examination:*** Early Diastolic murmur best heard over the 2nd aortic area and it increases by expiration.

➢ *When aortic regurgitation is severe it may lead to the following signs:*

| Peripheral Signs of AR which are due to ↑ pulse pressure | |
|---|---|
| Eponym | Sign |
| **DeMusset's sign** | Systolic head bobbing "nodding" |
| **Quincke's pulses** | Visible pulsations in the nail beds |
| **Corrigan's pulse** | Rapid-rising and rapid-collapsing carotid pulse |
| **Traube's sign** | Pistol shot sounds over the radial or femoral artery |
| **Duroziez's sign** | To-and-fro bruit over the femoral artery |
| **Muller's sign** | Systolic bobbing of the uvula eye ball |
| **Hill's sign** | Systolic BP in leg >20mmHg higher than in arm |

## *Investigation*

- **ECG:**
  - Left ventricular hypertrophy (usually) ± Ischemic changes
- **Chest X-ray** → May show Cardiomegaly due to LV dilatation
- **Echocardiography**: is the **Initial investigation of choice** because it Confirms the diagnosis **&** Assesses the severity of the regurgitation

## *Management*

- Treat arrhythmias and HF & HTN
- Prophylaxis for infective endocarditis & Prophylaxis for rheumatic fever [if <25yr]
- Asymptomatic pt → follow up by echo every 6-12 months.
- Afterload reduction (ACE inhibitors)→ ↓ regurgitation & ↑ forward cardiac output.
- **Surgery:**
  - Indications:
    - Symptomatic pt
    - Asymptomatic pt: LV dysfunction (LVEF < 55% or endsystolic LV diameter by echo > 55 mm) Operation done *before* development of CHF.
    - Type of Surgery: Repair or Replacement.

Valvuplasty ↘ valve replacement

# Tricuspid regurgitation

## *Causes*

1- **Infective endocarditis in drug abusers**
2- Right ventricular failure which leads to stretching of valve rings
3- Rheumatic heart disease
4- Congenital



## *Clinical picture*

It give the clinical picture of Right sided heart failure

*Examination*

- JVP → large **v** wave is seen in jugular veins during systole.
- Pulsatile liver: Systolic expansion of the liver frequently is present.

*Precordial examination*: Pansystolic murmur best heard over the tricuspid area with and it increases by inspiration.

## *Investigations*

**ECG:**

- May show Right ventricular hypertrophy

**Chest X-ray** → May show Cardiomegaly due to right ventricular dilatation
**Echocardiography:** is the **Initial investigation of choice** because it Confirms the diagnosis & Assesses the severity of the regurgitation

## *Management:*

- Intensive diuretic therapy when right-sided heart failure signs are present.
- In severe cases (in absence of severe pulmonary hypertension), surgical treatment consists of tricuspid annuloplasty (repair) or valve replacement.

**Austin Flint murmur**:. A low-pitched mid-diastolic rumble similar to that heard in mitral stenosis may be present in patients with **aortic regurgitation**. It's due to fluttering of anterior mitral valve due to the regurgitant flow.

**Carey-Coombs murmur**: A short mid-diastolic murmur is rarely heard during an episode of **acute rheumatic fever** and is due to enhanced flow through an edematous mitral valve.

**Graham-Steell murmur** is a high-pitched, early diastolic murmur that occurs in **Pulmonary regurgitation** (may be caused by pulmonary hypertension) & it's most prominent in the left parasternal region in the 2nd to 4th intercostal spaces.

**Carcinoid syndrome**: will cause Tricuspid or Pulmonary valve lesions

**SLE** will cause Mitral or Tricuspid valve lesions

| | | MS | MR | TR | AR | AS |
|---|---|---|---|---|---|---|
| **Inspection** [Pulsation] | | ± over Pulmn area<br>± over Lt. Parasternum | ± over Apical area<br>± over Lt. Parasternum | ± over epigastrium<br>± over Lt. Parasternum | ± over Apical area, Aortic area | ± Over the apical areas |
| **Palpation** | | | | | | |
| *Apical beat*<br>• **Site** | | Not displaced | ± **Displaced** down & laterally | ± Displaced laterally | ± **Displaced** down & laterally | Not displace |
| • **Character** | | Tapping | Sustained | Normal | Sustained | Forceful + Sustained |
| *Thrill* | | ± over mitral area | ± over mitral area | ± over tricuspid area | ± over aortic | ± over aortic area |
| *Left parasternal heave* | | ± | ± | ± | no | no |
| **Auscultation** | | | | | | |
| **S1** | | Loud | Soft | Normal | Normal | Normal |
| **S2** | | Normal or ↑ | Normal or ↑ | Normal or ↑ | Soft | Soft |
| **MURUR** | Site | **Mitral** | **Mitral** | Tricuspid | Aortic | Aortic |
| | Time | **Mid- diastolic** | **Pansystolic** | **Pansystolic** | **Early diastolic** | **Ejection Systolic** |
| | Radiation | No | To Axilla | No | No | To Carotid |
| | Inspiration | ↓ | ↓ | ↑ | ↓ | ↓ |
| Added sound | | Opening snap | S3 over mitral area | S3 over tricuspid area | S3 over mitral area | Ejection click |

*Causes of Stenosis:*
- Rheumatic fever [MS]
- Idiopathic calcification [AS]
- Congenital

*Causes of Regurgitation:*
- Rheumatic fever
- Infective endocarditis
- Heart failure [MR, TR]
- Congenital

*Investigations:*
1. ECG
2. CXR
3. Echo

*Complications:*
1- Arrhythmias mainly AF
2- Thrombo-embolisation
3- Of the left side → LVF
4- LVF→ Pulm. HTN → RVF
5- Of Right side→ RVF
6- RVF→ hepatic cirrhosis

*Treatment:*
- Explain to pt about his disease
- Rx arrhythmias & HF
- Prophylaxis for infective endocarditis
- Prophylaxis for rheumatic fever [if <25yrs]
- Asymptomatic pt → follow up by echo
- Surgery if pt becomes symptomatic
  - If stenosis→ balloon valvoplasty or replacement
  - If reguirge→ repair or replacement

# Infective Endocarditis

***Definition***: its infection of heart valves or other endocardial structures.
most commonly by gram bacteria

## *Epidemiology*

- Age: more in elderly.
- Gender: more in males.
- Mitral valve is the most affected valve, then aortic> tricuspid> pulmonary.

## *Risk factors:*

1. Any Valve disease Congenital [e.g. VSD, bicuspid aortic valve, & PDA] or Acquired [e.g. Rheumatic fever]
2. IV drug users —> staphylococcus
3. Prosthetic heart valves

*Classification of infective endocarditis*

- Acco. to type of valve
  - Native valve endocarditis [NVE]
  - Prosthetic valve endocarditis [PVE]
- Acco. to clinical picture
  - Acute infective endocarditis
  - Subacute infective endocarditis

## *Etiology*

| *Organism* | *Feature* | *C/P* |
|---|---|---|
| Streptococcus viridans | Most common cause for native valve endocarditis Dental abscess | **Subacute IE** |
| Staphylococcus epidermidis | Most common cause for Prosthetic valve endocarditis in the first year of surgery | Subacute IE |
| Staphylococcus aureus | **Commonest** cause in **IV drug abuser** & tricuspid valve is most involved Pulmonary | Acute IE |
| Enterococci | From GIT or Genitourinary system | Acute IE |
| *Streptococcus bovis* | Associated with colon polyps or cancer bowel | Subacute IE |
| Brucella ~~Gaffkemia~~ | H/O contact with goats or cattle & aortic valve is most involved | Subacute IE |
| Candida | Culture -ve endocarditis + large vegetation | Subacute IE |
| Q fever Coxiella | Culture -ve endocarditis +hepatosplenomegaly | Subacute IE |

## *Pathogenesis*

- Endothelial injury occurs → platelet-fibrin thrombus develop which become infected during transient bacteremia.
- Transient bacteremia may occur during dental, gastrointestinal, genitourinary or gynecological procedures; dental procedures are associated with highest risk. Transient bacteremia may occurs after tooth brushing ,eating, & bowel movements.
- Narrower the lesion, more is the risk[small VSD has higher risk than large VSD]

The **vegetation** is the prototypic lesion at the site of infection and it is composed of a mass of platelets + fibrin + microcolonies of organisms

***Clinical Presentation*** → Acute infective endocarditis
→ Subacute infective endocarditis

- ~~Acute infective endocarditis~~ it take
- It is caused by virulent organism & it can affect normal valves. most commonly staph & strept
- Presents with fever and rapid damage of heart valves → C/P of CHF with murmurs. [Heart block may occur when infection extends into conduction system].
- Metastatic infection are common. & It can progress to death within weeks.
- The systemic features of subacute infective endocarditis are **absent**

- Subacute infective endocarditis [SIE] Streptococcus viridans
  - **Subacute endocarditis** follows an indolent course, rarely causes metastatic infection, and progresses gradually unless complicated by an emboli.
  - **Constitutional symptoms** [Fever, Weight loss, Night sweating, & anorexia]
  - ~~**Systemic emboli**~~:
    1. Stroke (CNS embolism) → brain abscess
    2. Limb ischemia: acute limb pain.
    3. Renal embolism: flank pain and hematuria
    4. Splenic embolism: left upper quadrant or left shoulder pain
    5. Mesenteric embolism: abdominal pain and hematochezia.
    6. Coronary artery embolism: MI.
    7. Hand cutaneous embolism: **Janeway lesions** [Erythematous, painless lesions on the palms and soles]
    8. Conjunctival vessel embolism: Conjunctival hemorrhages
  - **Pulmonary emboli**: occurs in IV drug users with tricuspid or pulmonic valve endocarditis & presents with cough, hemoptysis, & pleuritic chest pain.
  - **Immune complex mediated symptoms**: The infected vegetation contains antigens that trigger an immune response→ production variety of autoantibodies (e.g. Rheumatoid Factor)→ Immune complex depositions which may cause extracardiac manifestations, but these classic signs are often absent:
    1. **Finger clubbing**
    2. **Splinter hemorrhage** of the nails
    3. **Roth's spots** by fundoscopy [Whitish, oval, lesions with surrounding hemorrhage]
    4. **Osler's nodes** [Tender, erythematosus lesions of the finger or toe pads]
    5. Glomerulonephritis [Red cells casts and proteinuria]
    6. Splenomegaly is found in 30-40% of pts
    7. Petechiae

**Prosthetic valve endocarditis**

- Infection usually affects the valve ring & may be subacute or acute endocarditis, depending on the virulence of the organism.
- Staphylococcus epidermidis [coagulase-negative staphylococci] is the commonest cause for Prosthetic valve endocarditis in the first year of surgery but after 1 year of valve surgery, it is caused by the same organisms native valve endocarditis.

## *Investigations*

1- Blood culture
- It is the **most important laboratory investigation** in diagnosis of endocarditis.
- Three sets of blood cultures should be taken before starting therapy, and there is no need to wait for episodes of pyrexia.
- Aseptic technique is essential and the risk of contaminants should be minimized by sampling from different venepuncture sites. An in-dwelling line should not be used to take cultures.

2- **Echocardiography:** Transthoracic echocardiography is the initial test of choice to see valvular vegetations & to measure valvular dysfunction. Although Transesophageal echo is more sensitive but negative TEE does not exclude IE

3- **ECG** may show conduction defect due to abscess involving the conducting system or MI

4- **CXR** may show abscesses due to septic emboli from right-sided endocarditis.

5- Routine laboratory test may reveal
- ↑ ESR and CRP the more sever the condition is
- CBC→ normocytic, normochromic anemia, ↑WBC and ↓Plt may be present.
- Urine analysis→ Proteinuria and microscopic hematuria is usually present

| Modified Dukes Criteria for diagnosis of infective endocarditis |
|---|
| **Major criteria**<br>1. Positive blood culture [ ≥ 2 positive cultures drawn> 12 hours apart OR all 3 cultures positive if drawn at least one hour apart OR a majority positive if 4 or more cultures are drawn]<br>2. Endocardial involvement<br>• Positive echocardiographic findings of vegetations<br>• New valvular regurgitation |
| **Minor criteria**<br>1. Predisposition: predisposing heart condition or injection drug use<br>2. Fever >38.0<br>3. Embolic phenomenon: major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hge, conjunctival hge, Janeway lesions<br>4. Immunologic phenomena: glomerulonephritis, Osler's nodes, Roth's spots, rheumatoid factor<br>5. Microbiologic evidence: positive blood culture but not meeting major criterion |
| • **Definite endocarditis:** 2 major, **OR** 1 major & 3 minor, **OR** 5 minor<br>• **Possible endocarditis:** 1 major and 1 minor, or 3 minor |

## *Treatment*

According to the presentation & type of valve involved Empirical treatment is started:

| Type | Empirical Antibiotic regimen |
|---|---|
| **Acute endocarditis** | |
| Native valve | Ampicillin + Flucloxacillin + Gentamicin |
| Prosthetic valve | Ampicillin + Ceftazidime + Gentamicin |
| **Subacute endocarditis** | Ampicillin + Gentamicin |

- Period of **4 to 6 weeks** of intravenous therapy is required.
- Recurrence of fever may indicate treatment failure, but may also result from hypersensitivity reactions to antibiotics.
- Monitoring treatment: **Serum CRP** is better than the ESR
- Surgical management: indications for valve replacement are:
  1- Fungal endocarditis.
  2- Congestive heart failure (CHF).
  3- Valve ring abscess.
  4- Failure to clear infection after a long course of antimicrobial therapy.

-Prosthetic valve
-Failure to clear embolization
-valve damage

## *Prophylaxis against infective endocarditis*

Used in pts undergoing dental, genitourinary, or gastrointestinal procedures.
In general, **Amoxicillin** 2 grams, or **Clindamycin** 600mg (if penicillin-allergic) given one hour before the procedure. In high risk pts undergoing genitourinary or gastrointestinal procedures add **Gentamicin** .

Patients at risk for IE:

1- Any prosthetic valve or arterial Dacron graft or Pacemaker
2- Prior infective endocarditis
3- All congenital heart disease except ASD
4- Any acquired valve defect [e.g. Rheumatic valve disease]
5- Mitral valve prolapse only if the valve is regurgitant
6- Hypertrophic obstructive cardiomyopathy

ASD & MVP without regurgitation are NOT risk factors for endocarditis

## *Prognosis*

Mortality rate is 15% in NVE, & 20-25% in PVE.
Factors that ↑ mortality: Age >65 yrs , Aortic valve infection, CHF, CNS involvement.

# Myocarditis flue like illness

***Definition***: Inflammation of the myocardium, it may be associated with pericarditis.

## *Etiology*

1. Viral infections is most common cause [**Coxsackie B**, adenovirus , influenza, **HIV**]
2. **Acute rheumatic fever**
3. Lyme disease (Borrelia burgdorferi)
4. Chaga's disease (Trypanosoma cruzi)
5. Toxins (e.g., **Cocaine**, **Doxorubicin**)

## *Clinical picture*

### *Symptoms*

- Viral myocarditis may be preceded by URI.
- Constitutional symptoms: Fever, fatigue.
- Chest pain
- Arrhythmias→ palpitations [It may be the cause of sudden death in young athlete]
- If LV dysfunction is present then it → symptoms of CHF.

### *Signs*

- Fever, & relative tachycardia.
- Pericardial friction rub if the pericardium is involved

– muffled heart sound & Apex beat

### *Investigations*

- *Lab:* CK-MB isoenzyme and cardiac troponins may be ~~elevated in absence~~ of MI, ESR is markedly raised, Elevated antiviral titers.
- *ECG* that may show transient ST elevation, **diffuse T wave inversions**, atrial and ventricular arrhythmias. Low voltage ECG
- *CXR* Cardiomegaly
- *Echocardiogram:* ↓ LV function; pericardial effusion may be present. ↓EF
- Endomyocardial biopsy if diagnosis is uncertain

## *Treatment*

- Strict bed Rest (because exercise may precipitate arrhythmias)
- Treat as CHF + Treat arrhythmias if present
- Immunosuppressive therapy (steroids and azathioprine) may be used.

IV immunoglobuline

***Prognosis***: In most cases the disease is self limiting, with excellent prognosis but some may progress to dilated cardiomyopathy.

# Cardiomyopathy

Dilated | Restrictive (Oblitrative) | Hypertrophic

## Hypertrophic Cardiomyopathy

***Definition***: Marked LV hypertrophy; often asymmetric [**affecting septum**], without underlying cause. Systole is normal, but ↑ LV stiffness → ↑ diastolic filling pressures [Diastolic HF]

### *Epidemiology*

- The most common form of cardiomyopathy
- The most common cause of sudden death in young athletes
- Autosomal dominant disorder in 60% of cases

### *Clinical picture*

#### *Symptoms*

- Arrhythmias [Sudden death may occur].
- Exertional Dyspnea or Angina or Syncope [due to outflow obstruction as in AS]
- C/P of CHF may occur.

#### *Signs*

- Carotid Pulse: Jerky
- Apex beat: Jerky heaving or double apical beat.
- Auscultation: a systolic murmur along left sternal border due to subaortic obstruction, murmur ↑ with Standing & Valsalva maneuver.

### *Investigations*

**CXR**: Mild to moderate cardiac shadow enlargement
**ECG**: LV hypertrophy with prominent "septal" Q waves in leads I, aVL, V5–6.
**ECHO**: LV hypertrophy, often with asymmetric septal hypertrophy.

### *Treatment*

- Strenuous exercise **should be** avoided.
- β-blockers and CCB reduce symptoms.
- Digoxin, Diuretics, and Vasodilators are *contraindicated*.
- Endocarditis antibiotic prophylaxis.
- Antiarrhythmic: amiodarone, may suppress atrial and ventricular arrhythmias.
- in pts refractory to medical therapy controlled septal infarction by ethanol injection into the septal artery or Surgical myectomy may be useful.
- Implantable automatic defibrillator (ICD) for pts with high-risk profile (e.g. FH of sudden death).
- Screening of first degree relatives is indicated

# Dilated cardiomyopathies

***Definition***: Symmetrically dilated left ventricle (LV) ± ventricle (RV) with poor systolic contractile function. [Systolic HF]

***Etiology***:

1- Idiopathic [most common]
2- Severe coronary disease or infarctions or chronic aortic/mitral regurgitation.
3- Previous myocarditis
4- Toxins (**Ethanol, Doxorubicin**)
5- Connective tissue disorders
6- Muscular dystrophies (Duchenne)
7- Beriberi (Thiamin deficiency)

***Clinical picture***: CHF + Arrhythmias [may → sudden death] + Thromboemolisms

***Investigations***

**CXR**: Moderate to marked cardiac shadow enlargement ± pleural effusion
**ECG**: ST-segment & T-wave abnormalities, Low voltage, Conduction defects [LBBB]
**ECHO**: LV and RV enlargement with globally impaired contraction. [unlike Regional wall motion abnormalities occurring in coronary artery diseases].

# Restrictive (Oblitrative) Cardiomyopathy

***Definition***: Increased myocardial stiffness which impairs ventricular relaxation; diastolic ventricular pressures are elevated. [Diastolic HF]

***Etiology***:

1- Infiltrative disease[Amyloidosis, Sarcoidosis, Hemochromatosis, Eosinophilic disorders]
2- Myocardial fibrosis [idiopathic]
3- Fabry's disease
4- Fibroelastosis

***Clinical picture***: CHF + Arrhythmias [may → sudden death] + Thromboemolisms
**CXR:** Mild cardiac shadow enlargement
**ECG:** ST-segment and T-wave abnormalities
**ECHO**: Increased ventricular thickness with speckled pattern in infiltrative disease.

***Treatment for Dilated & Restrictive cardiomyopathies***

- Standard therapy of CHF + **Anticoagulation**
- Antiarrhythmic drugs indicated only for symptomatic or sustained arrhythmias.

Dressler's syndrome

# Acute Pericarditis

***Definition:*** An acute inflammation of the pericardium

***Etiology:*** Most common causes are viral infections and Idiopathic.

- Idiopathic
- Infections (Viral : Coxsackie B virus, HIV, or Bacterial: TB) — late
- Acute MI (Dressler's syndrome or acute regional pericarditis) — early
- Metastatic neoplasm (Hodgkin & CA lung)
- Radiation therapy for tumor (up to 20 years earlier)
- Chronic renal failure = **Uremia**
- Connective tissue disease (Rheumatoid arthritis, SLE)
- Drug reaction (e.g., procainamide, hydralazine)

Myxedema (hypothyroidism)

*Symptoms*

- Acute pericarditis may be preceded by URI.
- Chest pain, characteristically sharp, pleuritic (related to respiration), & positional (relieved by leaning forward)
- Fever and palpitations are common.

*Signs*

- Tachycardia & pulse may be irregular if arrhythmia is present.
- Palpation: Apex beat may be impalpable.
- Auscultation: Muffled heart sound due to pericardial effusion ± Coarse **pericardial friction rub**, which is loudest with pt sitting forward.

*Investigations*

*ECG:* Diffuse ST elevation (concave upward) present in all leads except aVR and V1; PR segment depression may be present; *days* later (unlike acute MI) ST returns to baseline and T-wave inversion develops. Atrial premature beats and AF may appear.

| | ST-Segment Elevation | Evolution of ST and T Waves |
|---|---|---|
| Pericarditis | • Concave upward<br>• All leads except aVR and V1 | ST remains elevated for days after ST returns to baseline, T waves invert |
| Acute MI | • Convex upward (downward)<br>• ST ↑ over infracted region | T waves invert in hours while ST still elevated; Q wave |

*CXR:* Increased size of cardiac silhouette if large (>250 mL) pericardial effusion is present, with "**Water bottle**" configuration.

*Echocardiogram:* Most sensitive test to pericardial effusion which is commonly found

***Treatment***

- The disease resolves spontaneously over 3-10 days
- NSAIDs [Indomethacin] is enough usually but if *severe* pain ± prednisone
- Anticoagulants are relatively contraindicated due to risk of pericardial hemorrhage

***Complications***

- Inflammatory fluid collects → pericardial effusion; large effusions may → **Pericardial tamponade.**
- Chronically Inflammation → pericardial thickening & reduced pericardial compliance → **Pericardial constriction.**

# Cardiac Tamponade

Life-threatening emergency resulting from accumulation of pericardial fluid under pressure → impaired filling of cardiac chambers and decreased cardiac output. with normal EF

## *Etiology*

- Previous pericarditis (most common causes are metastatic tumor, uremia, acute MI, viral or idiopathic pericarditis).
- Cardiac trauma or myocardial infarction with left ventricular free wall rupture.

## *Pathophysiology*

- Normally, the pericardial space contains about 50 mL of fluid.
- Intrapericardial pressure = Intrathoracic pressure, When additional fluid enters the pericardial space, intrapericardial pressure rises. If fluid accumulates rapidly tamponade occurs with fluid accumulation of 200 mL. & if the fluid accumulates gradually, the pericardium may contain liters with only small rise in pressure .
- In diastole, ↑ intrapericardial pressure → elevation of diastolic pressure in all cardiac chambers (equilibration of pressures). The ↑ ventricular diastolic pressures impair ventricular filling → ↓ cardiac output and ↑ (JVP).

## *Clinical picture*

• Symptoms of pericarditis +

**Beck's Triad:** imp

1. Hypotension
2. Raised JVP
3. Soft or absent heart sounds

## *Signs*

- Signs of pericarditis [Muffled heart sounds ± impalpable apex beat]
- **Pulsus paradoxus** [decrease in LV volume during inspiration is due to (bulging of intraventricular septum from right to left) & (decreasing pulmonary venous return)]
- **Tachycardia**
- **Hypotension**
- ↑ JVP with rapid x-descent (rapid atrial filling after atrial contraction) and an absent y-descent (no passive filling of ventricle due to ↑ vent. diastolic pressure)
- If tamponade develops subacutely → RVF signs (LEE, hepatomegaly, and ascites)

## *Investigations*

- **ECG:** Low limb lead voltage, large effusions may cause **electrical alternans** (alternating size of QRS complex due to alternation of heart).
- **CXR:** Enlarged cardiac silhouette if large (>250 mL) effusion present. Flask shaped
- **Echocardiogram:** Swinging motion of heart within large effusion.
- **Cardiac Catheterization** Confirms diagnosis; shows equalization of diastolic pressures in all four chambers.

## *Treatment*

Immediate pericardiocentesis = Aspiration.

## Chronic Constrictive Pericarditis

It's a type of — in wich the pericardium becomes adhisive thick & thight that will lead to compretion on the heart

## Causes of pulses paradoxes

- acute sove Astma
- acute massive pulmonary embolism
- Sup. Vena caval obstruction
- cardiac Tamponad
- Chronic constrictive pericarditis

- You should suspect constrictive pericarditis when the patient has heart failure while the patient has asmal heart

# Constrictive Pericarditis

Constrictive pericarditis is the diffuse thickening of the pericardium in reaction to prior inflammation → rigid pericardium → impaired cardiac filling, elevation of systemic and pulmonary venous pressures, and decreased cardiac output.

## *Etiology*

It's due to healing and scar formation in some pts with previous pericarditis.
[Tuberculosis, Viral, Uremia, Neoplastic pericarditis]

## *Clinical picture*

***Symptoms:*** Symptoms of RVF; symptoms of LV failure uncommon.

## *Signs*

- Tachycardia
- Jugular venous distention (prominent y descent), which increases on inspiration (**Kussmaul's sign**)
- Sings of RVF (hepatomegaly, ascites, peripheral edema are common)
- Palpation→ apical pulse is reduced and may retract in systole (***Broadbent's sign***)
- Auscultation→ early, relatively high-pitched diastolic sound known as [**Pericardial knock**]. This sound occurs early in diastole as a result of the rapid cessation of ventricular filling as the pericardium is stretched to its limit.
- Pulsus paradoxus is present in ⅓ of pts.
- **Ascites precox**→ ascites that occurs earlier than peripheral edema

## *Investigations*

- **ECG** Low limb lead voltage; atrial arrhythmias are common.
- **CXR** Rim of pericardial calcification in up to 50% of pts.
- **Echocardiogram** Thickened pericardium, normal ventricular contraction; abrupt stop in ventricular filling in early diastole.
- **CT or MRI** is better than echocardiogram in showing pericardial thickness.
- **Cardiac Catheterization** Equalization of diastolic pressures in all chambers; Pts with constrictive pericarditis should be investigated for tuberculosis.

## *Treatment*

Surgical resection of the pericardium (**pericardiectomy**)

| | **Cardiac tamponade** | **Constrictive pericarditis** |
|---|---|---|
| **JVP** | Absent Y descent | X + Y present |
| **Pulsus paradoxus** | Present | Only in one third of pts |
| **Kussmaul's sign** | Rare | Present |
| **Investigation** | | Pericardial calcification on CXR |

systemic

It's an inflamatory, autoimmune disease which follow Group A B hymolytic infection after about 10 days to 2 weeks

R.F valve Rhough edge → calcification

Cobla positive → good week

Mayload to stenosis or regurge or both

symptoms of R.F

- dyspnea
- Syncope "Heart block"
- pericardial friction rub
- palpitation
- chest pain

★ Arthritis: - Swollen, Arrithmic, painfull joint
- it's one of the early appearing symptoms
- dramatically response to Aspirin

Carey Coomb murmur

# Rheumatic fever

***Definition*:** Acute rheumatic fever (RF) is an immune-mediated, inflammatory disease that result from untreated Group A β-hemolytic Streptococcal pharyngitis.

## *Epidemiology*

- More in underdeveloped countries, especially people living in overcrowded areas.
- **Age**: typically 5-15 year old. Preschool children
- **Gender**: Males = Females.

## *Pathophysiology*

- Group A β-hemolytic streptococcus (GABHS) which has M protein that look similar to some proteins in the heart (mimicry theory) → similar to myofibrilin
- Streptococcal skin are **not** associated with rheumatic fever.
- The pathognomonic lesion of rheumatic carditis is the Aschoff body.

## *Clinical picture*

All pts with RF have preceding streptococcal pharyngitis but it may be asymptomatic in 50% of cases. The symptoms of RF begin 2-3 weeks after the start of pharyngitis.

## *Major Manifestations*

1. **Carditis** more common in Pediatric age group
   - It occurs in 60% of pts and It affects all three layers of the heart endocardium, myocardium, and pericardium [Pancarditis].
   - RF most commonly affects mitral valve > aortic > tricuspid > pulmonic
   - Most pts with acute rheumatic carditis have a systolic murmur of **MR** and may have a low-pitched, apical, mid-diastolic murmur (Carey-Coombs murmur) resulting from flow across the inflamed valve. Some pt have AR.
   - Rheumatic myocarditis: presents as sinus tachycardia disproportionate to the degree of fever ± C/P of heart failure.
   - Rheumatic pericarditis →chest pain and audible **friction rub** and pericardial effusion. Tamponade is a rare.
2. **Arthritis**
   - It is the most common symptom of RF occurring in 75% of pts.
   - An asymmetric, non erosive, migratory polyarthritis mainly affecting large joints of extremities. Effusions are common, but heals without joint deformity.
3. **Chorea [Sydenham's chorea, St. Vitus' dance]** involuntary movement affecting the body proximal parts of
   - Chorea is seen in 20% of pts, its more in females and reflects inflammation of the basal ganglia.
   - It is characterized by purposeless, involuntary movements of the face & extremities, nervousness, explosive speech, emotional lability. Symptoms are absent during sleep and resolve spontaneously in 1-2 weeks.
   - Unlike the other symptoms, chorea appears 3-6 months after the pharyngitis.

Jones Criteria

4. **Erythema Marginatum :** Seen only in 5% of pts and it is an Erythematous, migratory, non-pruritis. The rash is serpiginous with raised margins & pale center. It is found in trunk and proximal extremities. "not the face" never
5. **Subcutaneous Nodules:** Seen in 3% of pts; Pea-sized, firm, painless, freely mobile nodules on the extensor surface of the elbows, knees, and wrists.

### *Minor Manifestations*

Fever, arthralgia (without arthritis), epistaxis, abdominal pain, and pleural effusion.

### *Late Manifestations*

About 50% of pts who have carditis during an episode of acute RF will eventually develop chronic rheumatic valvular disease, which is more common in ♀.
MR and AR may occur during the acute phase, or develop years later. But Valvular stenosis [MS or AS] is a late complication usually decades after the acute RF.

***Diagnosis:*** There is no definitive test or pathognomonic sign for acute RF and therefore the **Jones Criteria** is used for diagnosis.

| Major manifestations | Minor manifestations |
|---|---|
| 1. Carditis<br>2. Polyarthritis<br>3. Chorea<br>4. Erythema marginatum<br>5. Subcutaneous nodules | **Clinical**: previous history of R.F<br>1. Fever<br>2. Polyarthralgia<br>**Laboratory**:<br>3. Elevated ESR, CRP or WBC<br>4. ECG: prolonged P-R interval "element of heartblo |
| Supporting evidence of a preceding streptococcal infection within the last 45 days:<br>• Elevated or rising anti-streptolysin O or other streptococcal antibody, *or* ASO<br>• A positive throat culture, *or* Raised ASO titer<br>• Rapid antigen test for group A streptococcus, *or*<br>• Recent scarlet fever | |

MCQ

| 2003 WHO Criteria for the Diagnosis of Acute RF (Based on Revised Jones Criteria) | |
|---|---|
| **Diagnostic Categories** | **Criteria** |
| Primary episode of rheumatic fever | **Two major** or **one major and two minor** criteria + evidence of GABHS infection |
| Exception:<br>➢ If the pt has Rheumatic heart the Dx of recurrent attack needs 2 minor criteria + evidence of GABHS.<br>➢ Rheumatic chorea [Other major criteria or evidence of GABHS not required]<br>➢ Insidious onset rheumatic carditis [Other major criteria or evidence of GABHS not required] | |

## *Treatment of acute rheumatic fever*

- Admission and bed rest. Pain killer
- All pts should receive 10-day course of penicillin-V (erythromycin if penicillin allergic) 1.2 IO-IM
- Arthritis usually responds well to high-dose Aspirin [Dramatic response]
- Patients with significant carditis, and those who do not respond an adequate dose of aspirin, are treated with steroids.

## *Prevention*

- Intramuscular benzathine penicillin (1.2 million units every 3-4 weeks) or oral penicillin VK (250 milligrams twice daily).
- Prophylaxis is recommended for at least 10 years after the most recent episode of acute RF, and generally until age 25-40 according to the pt.

# Hypertension

## *Definition*

The level of blood pressure above which treatment do more good than harm.

- In normal pt the level is **140/90 mmHg**
- In pt with DM, CRF the level is **130/80 mmHg**

## *Epidemiology*

Age→ ↑ with age
Gender→ male > female
Race→ more in Black

**Q.** What are the factors responsible for blood pressure in our body ?

1- The degree of contraction of the resistance bl. vessels (the arterioles).
2- Total body sodium[regulated by Aldesterone & Kidney]

BP = Cardiac output X Total peripheral resistance

## *Pathophysiology*

- **Essential = Primary HTN (95% of pts):** Unknown cause but a combination of **Genetic factors**[1- Black 2- Males] & **Environmental factors** [ 1- ↑ Salt intake 2- Alcohol 3- Obesity 4- ↓ exercise 5- DM2 (50% of pt are HTN) 6-IUGR.] Stress?
- **Secondary HTN** (5% of pts): HTN is 2nd ry to diagnosable disease.

## Secondary HTN

| Renal | Endocrine | Drugs | Miscellaneous |
|---|---|---|---|
| 1. Renal artery stenosis | 1-↑ or ↓ Thyroidism | 1-Corticosteroids | 1- Coarctation of aorta |
| 2. Fibromuscular dysplasia | 2-↑ or ↓ Parathyroidism | 2-NSAID | 2-Vasculitis |
| 3. Glumerulonephritis | 3-Pheochromocytoma | 3-Oral CCP | 3- ↑ ICP |
| 3-Polycystic kidney | 4-Cushing disease | 4-Erythropoietin | 4-Carcioniod synd. |
| 4-Chronic pyelonephritis | 5-Pregnancy | 5-Cyclosporin | 5- Polycythemia |
| | | β agonist | |

## *Ideal measurement of BP*

1. Put the pt right arm at the level of the heart. And remove cloths from the arm.
2. Select an appropriate cuff size which should cover 2/3 of arm length.
3. Estimate the systolic bp by palpating the brachial or radial artery & inflating the cuff until you can no longer feel it. [This to exclude auscultatory gap].
4. Inflate the cuff to 20-30 mmHg more than the estimated systolic bp.
5. Place the stethoscope over the brachial artery pulse, and don't touch the cuff.
6. Reduce the pressure in the cuff at a rate of 2-3 mmHg/sec.
7. The first Korotkov sounds indicate the systolic bp.
8. The muffling & disappearance of the Korotkov sounds indicate the diastotic bp.
9. Measure the BP in the other arm

| Definition of Hypertension | | |
|---|---|---|
| **Category** | **Systolic BP (mmHg)** | **Diastolic BP (mmHg)** |
| **Hypertension** | | |
| Grade 1 (mild) | 140-159 | 90-99 |
| Grade 2 (moderate) | 160-179 | 100-109 |
| Grade 3 (severe) | ≥180 | ≥110 |
| **Isolated systolic HTN** | | |
| Grade 1 | 140-159 | < 90 |
| Grade 2 | ≥160 | < 90 |

## *Investigations*

Note: To diagnose hypertension the pt must have 2 raised results

*In all pts: [aim to detect target organ damage and assessment of risk factors]*

- Urinalysis
- Blood urea, electrolytes and creatinine
- Blood glucose
- Lipid profile.
- 12-lead ECG (left ventricular hypertrophy, coronary artery disease)

*In selected pts:*

- Further Investigations are done when 2ndry HTN is suspected as in the following:
    1. Age < 20 years or >50 years.
    2. Pts without a family history of hypertension.
    3. Pts with hypertension refractory to medical therapy.

## *Clinical picture*

Hypertension is Asymptomatic disease and pt present with its complications.
↑ Systolic BP is as significant as ↑ Diastolic BP as a risk factor for CVS & renal diseases.

### Target Organ Damage

**Heart**
- Left ventricular hypertrophy → cardiomyopathy → **Heart failure**
- **Coronary artery disease** (Angina, MI) atherosclerosis
- Atrial fibrillation
- Aortic dissection ↑risk of acquired aneurysm

**Brain**
- TIA [transient ischemic attacks] CVA "cerebral vascular attack"
- Hemorrhagic stroke
- Subarachnoid hemorrhage [Bouchard's aneurysm]
- Hypertensive Encephalopathy [Headache, confusion, coma]

**Kidney**
- Proteinuria
- Renal failure
- Acute glomerulonephritis hematuria

**Eye** → Retinopathy
- Grade1: Arteriolar thickening [Silver wiring]
- Grade 2: Atrioveous crossing (nipping)
- Grade 3: Retinal ischemia (Flame-shaped Hge & Cotton wool exudate)
- Grade 4: Papilledema

**Peripheral arteries** → Claudication dilated & tortous arteries

## *Treatment*

- Goals of therapy is to reduce BP to < 140/90 mmHg in normal pts and to < 130/80 mmHg in pt with DM, CRF.

- Non-pharmacologic measures
  1. Sodium restriction < 2 g/day.
  2. Weight reduction in obese.
  3. ↓ Alcohol intake. [Alcohol ↑ action of catecholamines]

➢ Pharmacologic measures **ABCD**

**Angiotensin-converting enzyme (ACE) inhibitors** [e.g. Captopril, Enalopril, Lisinopril]

*Pharmacodynamics*

- ACE inhibitors mainly act by ↓ afterload through the reduction of AngII.
- ACEI reduces Salt & water retention by ↓ aldesterone.

*Side effects*:

5. Most common SE is cough, it occurs 10-15% of pts, it is due to accumulation of bradykinins which normally is converted to kinin by the ACE enzyme.
6. First dose hypotension
7. Hyperkalemia
8. Rare leukopenia, rash, and angioedema.

*Absolute contraindications* for ACEI & Angiotensin receptor blockers

1. Pregnancy
2. Hyperkalemia serum K > 5.5
3. Bilateral renal artery stenosis
4. Advanced renal failure (e.g., creatinine > 3 mg/dL)

Angiotensin receptor blockers [Losartan, Valsartan] is used when pt cannot tolerate ACEI due to the cough

**β-Blockers**

*Pharmacodynamics:* ↓ cardiac output and renin release.

*Agents:*

- Metoprolol, Atenolol [selective β1 blockers]
- Carvedilol, Labetalol [αβ-blocker]

*Contraindication*: See Heart failure sheet.

**Calcium channel antagonists**

*Pharmacodynamics*: arterial VD → total peripheral resistance → ↓ BP.

Agents:

- Dihydropyridines (nifedipine, nicardipine, amlodipine)
- Non-dihydropyridines (diltiazem, verapamil)

Side effects:

1. Headache
2. Flushing
3. Peripheral edema
4. Constipation
5. Verapamil & diltiazem have –ve inotropic actions on the heart may ↑ symptoms in pt with CHF.

## Diuretics

*Pharmacodynamics*: ↓ extracellular fluid volume + Vasodilator effects.

*Agents*

- **Loop diuretic** (Furosemide, Bumetanide) is the first line treatment [mechanism of action → inhibition of reabsorption of $Na^+$, $K^+$, 2 $Cl^-$ pump in the thick ascending limb of Henle's loop]
- **Thiazides** (Hydrochlorothiazide, Chlorthalidone, Chlorothiazide, Metolazone) [mechanism of action → reduce the reabsorption of $Na^+$ & $Cl^-$ in the distal convoluted tubule]

*Side effects:*

1- Volume depletion and hyponatremia
2- ↓ hypokalemia with Furosemide or Thiazides Metabolic alkalosis
3- Thiazide: Hyperuricemia → Gout, Hyperlipidemia, Hyperglycemia → DM, Hypercalcemia, Erectile dysfunction.

- **Aldesterone antagonist** [Spironolactone and eplerenone] Aldosterone receptor antagonists, Side effects: [Hyperkalemia ,Gynecomastia]

## *Other drugs:*

- $\alpha_1$-adrenoceptor antagonists such as prazosin and doxazosin
- Direct vasodilators: hydralazine and minoxidil. SE: first-dose & postural hypotension, headache, fluid retention. Minoxidil→ ↑ facial hair (avoid in females)
- Centrally acting drugs e.g. methyldopa and clonidine.

## Q. What Class should be used?

- In > 55 years and black: **C or D**
- In < 55 year: **A or B**
- Diuretics Thiazide is first line therapy of choice.
- If the pt has a coexisting medical conditions the choice may vary:
  - Coexisting CAD or compensated heart failure → β blockers because ↓ mortality.
  - Diabetes → ACE
  - Benign prostatic hypertrophy → α-blockers

## *Hypertensive crisis*

Group of syndromes associated with High BP >230/140 mmHg

- **Hypertensive urgency** = Severe HTN without acute target organ damage.
  - Rx: Oral antihypertensive drugs.

- **Hypertensive emergency**= Severe HTN with **Symptoms** of acute target organ damage.

**Symptoms**
- Headache, Confusion, Coma → Hypertensive encephalopathy
- Dyspnea → Heart failure
- Visual disturbance → Retinopathy
- Acute renal failure → Kidney

Rx: Parenteral [IV] antihypertensive:
- Labetalol IV or IM
- Glyceryl trinitrate IV
- Hydralazine IM
- Sodium nitroprusside IV

NOTE: **Don't ↓ BP rapidly** because this will → ischemic complications [ Ischemic stroke, MI, Blindness]

- **Malignant (Accelerated) HTN**: Severe Retinopathy (grade 3 or 4) ± other acute target organ damage.
  - Rare condition & called malignant because → death in months.